U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 09 March 2004

IN THE MATTER OF:

AGNES SCHUTT (widow of and on behalf of deceased miner BENEDICT SCHUTT),
Claimant,

v. Case No.: 2001-BLA-1010

KNIFE RIVER COAL CO.,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest.

APPEARANCES: David C. Thompson, Esq.
For the Claimant

William S. Mattingly, Esq.
For the Employer

BEFORE: Thomas M. Burke
Associate Chief Administrative Law Judge

DECISION AND ORDER AWARDING MINER'S AND SURVIVOR'S BENEFITS

This case arises from a claim for benefits filed under the Black Lung Benefits Act, as amended, at 30 U.S.C. § 901 *et seq.* ("Act"), and the implementing regulations thereunder at 20 C.F.R. Parts 718 and 725. A hearing was held in Bismarck, North Dakota on October 21, 2003. The decision in this matter is based upon the testimony of witnesses at the hearing, all documentary evidence admitted into the record at the hearing, and the post-hearing arguments of the parties.

I
Overview of the Black Lung Program

The Black Lung Benefits Act is designed to compensate those miners or survivors of miners who acquired pneumoconiosis, commonly referred to as "black lung disease," while

working in the Nation's coal mines. Entitlement is not automatic, nor does it serve as a pension or retirement program for coal miners or their survivors. Rather, those miners who have worked in or around mines and have inhaled coal mine dust over a period of time, may contract black lung disease. This disease may eventually render the miner totally disabled or contribute to his death.

II
Procedural History

1. On February 17, 1992, the miner filed a claim for benefits. *Director's Exhibit (Dx.)* 1.

2. By letter dated July 27, 1992, the district director proposed to deny benefits on grounds that the miner did not establish that he was totally disabled or that any potential disability was due to coal workers' pneumoconiosis. *Dx.* 13.

3. On August 6, 1992, Claimant requested a hearing. *Dx.* 14. By letter filed on December 28, 1992, the miner submitted additional evidence and stated that he would like a "modification." *Dx.* 17.

4. On July 6, 1993, the district director issued a proposed decision that the miner was entitled to benefits. *Dx.* 31.

5. On October 26, 1993, the miner's claim was referred to this office for adjudication. *Dx.* 34.

6. A *Certificate of Death* establishes that the miner died on June 2, 1997. *Dx.* 42.

7. By *Order* dated November 25, 1997, the undersigned remanded the case on Employer's motion for consolidation given that the miner passed away and Mrs. Schutt intended to file a claim for survivor's benefits. *Dx.* 64.

8. On March 9, 1998, Claimant filed for survivor's benefits. *Dx.* 72.

9. On March 12, 2001, the district director proposed to deny survivor's benefits on the grounds that Claimant did not establish that coal workers' pneumoconiosis caused the miner's death. *Dx.* 85.

10. By letter dated May 11, 2001, Claimant requested a hearing. *Dx.* 86.

11. On July 2, 2001, both claims were forwarded to this Office for adjudication. *Dx.* 91.

III
Issues Presented for Adjudication

The CM-1025 lists several issues being contested by Employer. *Dx.* 91. However, at the hearing Employer withdrew its controversion of many issues. Specifically, Employer stated that issues 1, 2, and 3 were no longer at issue. *Hearing Transcript (Tr.)* at 8. Employer further

stipulated to at least 28 years of coal mine employment. *Tr.* at 8-9, 11. Moreover, Employer agreed that the miner had one dependent, his wife Agnes, for purposes of augmentation of benefits. *Tr.* at 8. Finally, Employer asserted that the issue of proper designation of the responsible operator was no longer at issue. *Tr.* at 9. In its post-hearing brief, Employer states that Claimant has not remarried and she is an "eligible" survivor. *Id.* at 3.

The issues remaining for adjudication are as follows in the miner's and survivor's claims: (1) whether the miner suffered from pneumoconiosis; (2) whether the pneumoconiosis arose out of coal mine employment; (3) whether the miner was totally disabled; (4) whether the miner's total disability arose in part from his coal workers' pneumoconiosis; and (5) whether the miner's death was hastened by coal workers' pneumoconiosis.

IV
The Standard for Entitlement

Because the miner's claim was filed in February 1992 and the survivor's claim was filed in March of 1998, they are governed by the regulations at 20 C.F.R. Part 718 (2001).[1] Moreover, under § 718.205, where there is no miner's claim filed prior to January 1, 1982 resulting in entitlement to benefits, then a survivor who files a claim after January 1, 1982, as in this case, is entitled to benefits only upon demonstrating that the miner died due to pneumoconiosis.[2] 20 C.F.R. § 718.205 (2001). Specifically, Claimant bears the burden of establishing each of the following elements by a preponderance of the evidence: (1) that the miner suffered from pneumoconiosis; (2) his pneumoconiosis arose out of coal mine employment; (3) that the miner was totally disabled; (4) his total disability arose, at least in part, out of coal dust exposure; and (5) that he died due to pneumoconiosis. *See Gee v. W.G. Moore & Sons*, 9 B.L.R. 1-4 (1986) (en banc); *Baumgartner v. Director, OWCP*, 9 B.L.R. 1-65 (1986)(en banc). Evidence which is in equipoise is insufficient to sustain Claimant's burden in this regard. *Director, OWCP v. Greenwich Collieries,* 114 S. Ct. 2251 (1994), *aff'g sub. nom. Greenwich Collieries v. Director, OWCP*, 990 F.2d 730 (3rd Cir. 1993).

V
Existence of Pneumoconiosis and its Etiology

Under the amended regulations, "pneumoconiosis" is defined to include both clinical and legal pneumoconiosis:

> (a) For the purpose of the Act, "pneumoconiosis" means a "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments,

[1] As the miner engaged in coal mine employment in the State of North Dakota, appellate jurisdiction of this matter lies with the Eighth Circuit Court of Appeals. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989)(en banc).

[2] Because the survivor's claim was not filed prior to June 30, 1982, the presumption contained at 20 C.F.R. § 718.306 is inapplicable and will not be discussed further.

arising out of coal mine employment." This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis.

> (1) Clinical Pneumoconiosis. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition cause by dust exposure in coal mine employment. The definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.
>
> (2) Legal Pneumoconiosis. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

(b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 C.F.R. § 718.201 (2001). Moreover, the regulations at 20 C.F.R. § 718.203(b) (2001) provide that, if a miner suffers from pneumoconiosis and has engaged in coal mine employment for ten years or more, as in this case, there is a rebuttable presumption that the pneumoconiosis arose out of such employment.

The existence of pneumoconiosis may be established by any one or more of the following methods: (1) chest x-rays; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by a physician exercising sound medical judgment based on objective medical evidence. 20 C.F.R. § 718.202(a) (2001).

Summary of the chest x-ray evidence

The regulation at 20 C.F.R. § 718.202(a)(1) (2001) requires that "where two or more X-ray reports are in conflict, in evaluating such X-ray reports consideration shall be given to the radiological qualifications of the physicians interpreting such X-rays."[3] In this vein, the Board

[3] A"B-reader" (B) is a physician, but not necessarily a radiologist, who successfully completed an examination in interpreting x-ray studies conducted by, or on behalf of, the Appalachian Laboratory for Occupational Safety and

has held that it is proper to accord greater weight to the interpretation of a B-reader or Board-certified radiologist over that of a physician without these specialized qualifications. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Allen v. Riley Hall Coal Co.*, 6 B.L.R. 1-376 (1983). Moreover, an interpretation by a dually-qualified B-reader and Board-certified radiologist may be accorded greater weight than that of a B-reader. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Sheckler v. Clinchfield Coal Co.*, 7 B.L.R. 1-128 (1984). The following chest x-ray evidence is in the record:[4]

Exhibit Number	Physician/ Radiological Qualifications	Study date/ Reading date	Film Quality	Reading
Dx. 57	Repsher B	10-27-86 12-22-96	1	--
Dx. 28	Fino B	10-27-86 11-26-93	1	–; chronic infiltrate and scarring in left lower lung; emphysema or hyperaeration suggested
Dx. 28	Fino B	11-03-86 11-09-93	1	–; bilateral pleural thickening
Dx. 28	Wheeler B, BCR	11-03-86 09-03-93	1	–; moderate emphysema, minimal linear fibrosis, pleural fibrosis
Dx. 28	Kim B, BCR	11-03-86 08-29-93	1	–; chronic obstructive pulmonary disease
Dx. 57	Repsher B	11-03-86 12-22-96	1	--
Dx. 28	Eisner B, BCR	11-03-86 08-25-93	1	–; linear lung markings, fibrosis

Health (ALOSH). A designation of "Board-certified" (BCR) denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology or the American Osteopathic Association. An "A-reader" is a physician, but not necessarily a radiologist, who submitted six x-ray studies of his or her clients to ALOSH of which two studies are interpreted as positive for the existence of pneumoconiosis, two studies are negative, and two studies demonstrate complicated pneumoconiosis.

It is noted that Dr. Guidice, whose radiological qualifications are unknown, reviewed two studies on January 7, 1997. *Dx.* 58. The dates of the studies reviewed are illegible. Although the studies were not classified in accordance with the ILO/UICC classifcation system, he found the presence of pneumoconiosis, pleural asbestosis, and parenchymal asbestosis. Dr. Guidice's interpretations are entitled to little weight because his radiological qualifications, if any, are unknown. *Stanley v. Director, OWCP*, 7 B.L.R. 1-386 (1984).

[4] A "--" under the *Reading* column indicates that the physician did not interpret the study in accordance with the ILO-U/C classification system as required at 20 C.F.R. §§ 718.102 and 718.202(a)(1) (2001

Dx. 28	Scott B, BCR	11-03-86 08-09-93	1	–; scattered non-specific linear fibrosis
Dx. 28	Fino B	07-17-87 11-16-93	1	–
Dx. 28	Wheeler B, BCR	07-17-87 09-21-93	1	–; no evidence of silicosis or coal workers' pneumoconiosis
Dx. 28	Eisner B, BCR	07-17-87 08-25-93	1	–; linear markings, emphysema
Dx. 28	Scott B, BCR	07-17-87 08-09-93	1	–; scattered non-specific linear fibrosis
Dx. 28	Kim B, BCR	07-17-87 08-27-93	1	–; chronic obstructive pulmonary disease
Dx. 57	Repsher B	07-17-87 12-22-96	1	--
Dx. 28	Fino B	06-07-89 11-26-93	1	--
Dx. 57	Repsher B	06-07-89 12-22-96	1	--
Dx. 28	Fino B	10-11-90 11-16-93	1	–
Dx. 28	Wheeler B, BCR	10-11-90 09-21-93	2	–; no evidence of silicosis or coal workers' pneumoconiosis
Dx. 28	Scott B, BCR	10-11-90 08-09-93	1	–; minimal scattered non-specific linear fibrosis
Dx. 28	Kim B, BCR	10-11-90 08-27-93	1	–; chronic obstructive pulmonary disease
Dx. 28	Eisner B, BCR	10-11-90 08-25-93	2	–
Dx. 28	Fino B	10-24-90 11-26-93	1	–

Dx. 57	Repsher B	10-24-90 12-22-96	1	--
Dx. 28	Fino B	07-17-91 11-16-93	1	–
Dx. 29	Wiot B, BCR	07-17-91 03-15-94	1	–; no coal workers' pneumoconiosis, extensive pleural disease
Dx. 57	Repsher B	07-17-91 12-22-96	1	--
Dx. 29	Spitz B, BCR	07-17-91 03-22-94	1	–; no evidence of coal workers' pneumoconiosis, pleural disease consistent with pneumoconiosis, asbestos exposure; chronic obstructive pulmonary disease
Dx. 58	Powers B, BCR	07-17-91 01-08-97	2	1/2
Dx. 28	Wiot B, BCR	03-17-92 06-02-93	1	–; no coal workers' pneumoconiosis
Dx. 28	Shipley B, BCR	03-17-92 06-09-93	3	–; no coal workers' pneumoconiosis
Dx. 28	Spitz B, BCR	03-17-92 06-14-93	1	--
Dx. 28	Wheeler B, BCR	03-17-92 04-13-93	1	0/1; no evidence of silicosis or coal workers' pneumoconiosis
Dx. 57	Scott B, BCR	03-17-92 04-13-93	1	–; asbestos exposure cannot be excluded
Dx. 57	Repsher B	03-17-92 12-22-96	1	--
Dx. 28	Meseroll B, BCR	03-17-92 05-13-92	2	1/1

Dx. 28	O'Keefe radiologist	03-17-92 03-17-92	readable	–; chronic obstructive pulmonary disease, interstitial fibrosis and emphysema
Dx. 29	Spitz B, BCR	11-30-93 03-28-94	1	–; no evidence of coal workers' pneumoconiosis; pleural disease consistent with previous asbestos exposure; chronic obstructive pulmonary disease
Dx. 29	O'Keefe radiologist	11-30-93 12-01-93	readable	–; chronic obstructive pulmonary disease; chronic interstitial fibrosis
Dx. 29	Wiot B, BCR	11-30-93 03-26-94	1	–; no evidence of coal workers' pneumoconiosis; extensive pleural disease
Dx. 57	Wheeler B, BCR	11-30-93 12-22-94	1	–; moderate emphysema, linear and pleural fibrosis, no evidence of silicosis or coal workers' pneumoconiosis
Dx. 57	Fino B	11-30-93 01-24-95	1	0/0
Dx. 57	Repsher B	11-30-93 12-22-96	1	--
Dx. 35	Scott B, BCR	11-30-93 12-22-94	1	–; no evidence of silicosis or coal workers' pneumoconiosis; emphysema; non-specific linear fibrosis, possible asbestos plaques
Dx. 61	Wiot B, BCR	12-16-93 03-10-94	1	–; no coal workers' pneumoconiosis
Dx. 58	Powers B, BCR	12-16-93 01-18-97	2	1/2; lower lung zones

Dx. 35	Wheeler B, BCR	12-16-93 12-22-94	1	–; no evidence of silicosis or coal workers' pneumoconiosis; minimal linear fibrosis
Dx. 35	Scott B, BCR	12-16-93 12-22-94	1	–; emphysema, linear non-specific fibrosis or discoid atlectasis; possible asbestos plaques
Dx. 29	Podoll B, BCR	12-16-93 12-16-93	readable	–; emphysema and interstitial fibrosis unchanged from 11-30-93
Dx. 29	Fino B	12-16-93 02-21-94	1	1/1; no coal workers' pneumoconiosis; possible asbestosis or severe pneumonia
Dx. 57	Repsher B	12-16-93 12-22-96	1	--
Dx. 57	Repsher B	07-18-94 12-22-96	1	0/1
Dx. 53	Castle B	07-18-94 05-24-95	1	--
Dx. 57	Wiot B, BCR	02-07-95 05-31-95	1	–; no coal workers' pneumoconiosis; extensive bilateral pleural disease
Dx. 47	Lee B, BCR	02-07-95 03-26-95	2	2/2
Dx. 79	Kimmel radiologist	02-07-95 02-08-95	readable	–; obstructive lung disease; pulmonary arterial hypertension; mild vascular congestion with fluid and thickening; lateral pleural thickening which may be seen with asbestos exposure

Dx. 79	Young radiologist	07-13-95 07-13-95	readable	–; pulmonary emphysema; probable minimal left fluid effusion; minimal congestion in lower lungs
Dx. 62	Winjum radiologist	12-17-96 12-17-96	readable	–; moderate changes of congestive heart failure; small bilateral pleural effusions
Ex. 9	Wiot B, BCR	03-10-97 07-17-03	1	–; no coal workers' pneumoconiosis
Ex. 9	Wiot B, BCR	05-06-97 07-19-03	1	–; no coal workers' pneumoconiosis
Dx. 62	Hill radiologist	05-06-97 05-06-97	readable	–; pulmonary edema developed since 4-14-97; underlying fibrotic reaction in mid and lower lungs with some pleural thickening
Dx. 62	Stone radiologist	05-07-97 05-08-97	readable	–; heart mildly enlarged; diffuse interstitial thickening with pleural thickening bilaterally

Dr. Jerome F. Wiot was deposed with regard to his qualifications and interpretations on January 13, 1997. *Dx.* 60. He testified that he is a physician and diagnostic radiologist. *Dx.* 60 at 4. He is board-certified in radiology and serves as Professor and Chairman of Radiology at the University of Cincinnati, Director of Radiology at Cincinnati Hospitals, Cincinnati Children's Hospital, and the Cincinnati VA Hospital. *Dx.* 60 at 5. Dr. Wiot was one of the original C-readers and is a member of the NIOSH/ALOSH task force to develop the ILO/UICC classification system and he had "to do . . . teaching of individuals about coal workers' pneumoconiosis and how to use the classification system." *Dx.* 60 at 6, 8-9. He currently serves as Chair of the task force. *Dx.* 60 at 6.

Dr. Wiot stated that coal workers' pneumoconiosis starts at the top of the lungs and works downward. *Dx.* 60 at 20. Opacities are usually rounded, but may be irregularly shaped. *Dx.* 60 at 20. Moreover, the disease usually forms eggshell calcifications. *Dx.* 60 at 21.

Dr. Wiot concluded that the miner did not have coal workers' pneumoconiosis, but "[h]e has extensive pleural disease, greater on the left than on the right, but pleural disease is not a manifestation of coal dust exposure, and there's no findings to suggest coal workers'

pneumoconiosis." *Dx.* 60 at 25. Dr. Wiot found no changes in the x-rays that he reviewed over time. *Dx.* 60 at 26. He stated the following:

> [T]he upper lung fields on Mr. Schutt are perfectly clear, there's absolutely nothing to be seen. In fact, the whole right lung field where we can really get a good look at the lung is perfectly okay. Now, Mr. Schutt has pleural disease along the right lateral chest wall, he's got a so-called blunted angle and he's got pleural disease extending along the right lateral chest wall.
>
> On the left side, he's got also extensive pleural disease, but it's thick enough that it causes significant changes when we look at the PA film overlying the lung, so you can't really on the PA film, and that's what we work with as far as the ILO system is concerned, we can't evaluate the lower lungs on the lung field on the left because of the extensive pleural disease.
>
> . . .
>
> But he has this extensive pleural disease . . . this is most likely going to be an old inflammatory basis, but it's not in any way related to coal dust exposure.

Dx. 60 at 27-28. Dr. Wiot opined that the miner's pleural disease could be caused by old pneumonia or tuberculosis. *Dx.* 60 at 29.

On cross-examination, Dr. Wiot clarified that he saw no changes on the 1991 and 1993 films. *Dx.* 60 at 35. He conceded that pathology is the "gold standard" for diagnosing lung disease stating that "[y]ou can have microscopic evidence of asbestosis and not have radiographic evidence." *Dx.* 60 at 45. Dr. Wiot stated that he would have marked the box that pleural thickening was related to pneumoconiosis if he had known about the miner's asbestos exposure. *Dx.* 60 at 52-53.

Discussion of chest x-ray evidence

Upon review of the chest x-ray evidence, a majority of interpretations indicate the presence of fibrosis and pleural thickening, but they do not diagnose Category 1 pneumoconiosis or greater. In particular, studies dated October 27, 1986, November 3, 1986, July 17, 1987, June 7, 1989, and October 11 and 24, 1990 do not demonstrate the presence of pneumoconiosis.

Although a July 17, 1991 study was interpreted as Category 1/2 by a dually-qualified physician, two dually-qualified physicians and two B-readers concluded that it did not support a finding of pneumoconiosis. On balance, this study does not carry Claimant's burden under § 718.201(a) of the regulations.

A March 17, 1992 study was interpreted as positive for the presence of pneumoconiosis by a dually-qualified physician. However, this interpretation is outweighed by the negative findings of five dually-qualified physicians and a B-reader.

The November 30, 1993 study was interpreted negatively by four dually-qualified physicians and two B-readers. Moreover, while the December 16, 1993 study was interpreted as positive by a B-reader, five dually-qualified physicians and another B-reader concluded that it was negative. As a result, this study does not support a finding of the disease.

A study dated July 18, 1994 was interpreted as negative for the disease. In addition, while one dually-qualified physician interpreted the February 7, 1995 study as showing Category 2 pneumoconiosis, another dually-qualified physician, Dr. Wiot, concluded that the study did not support a finding of the disease. The record establishes that Dr. Wiot was one of the original C-readers and helped develop the ILO-UICC classification system. He conducts annual training for NIOSH on proper interpretation of x-ray studies under the system. As a result, his interpretation carries the greatest weight.

The remaining studies of record, dated July 13, 1995, December 17, 1996, March 10, 1997, and May 6 and 7, 1997 were not interpreted as positive for the presence of pneumoconiosis.

Consequently, the weight of the chest x-ray evidence does not support a finding of pneumoconiosis. Claimant has not sustained her burden under 20 C.F.R. § 718.201(a)(1) (2001) of the regulations.

Summary of the autopsy and medical opinion reports

Claimant may also establish that the miner suffered from the disease through well-reasoned, well-documented autopsy or medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See Hoffman v. B&G Construction Co.*, 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984).

A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra*. Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder-of-fact to decide. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(en banc). Moreover, statutory pneumoconiosis is established by well-reasoned medical reports that support a finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP*, 11 B.L.R. 1-135 (1988). The following medical reports were admitted as evidence in this record:

DR. SAMUEL SPAGNOLO

Dr. Spagnolo reviewed certain medical records and issued his first report on March 17, 1997. *Employer's Exhibit (Ex.)* 1. He noted that the miner complained of chest tightness and shortness of breath on exertion. Chest x-rays were interpreted by several "renowned radiologists including Dr. Wiot" as negative for the presence of pneumoconiosis. He concluded that the

miner did not suffer from coal workers' pneumoconiosis "primarily based upon the lack of chest x-ray findings consistent with coal workers' pneumoconiosis or physiologic evidence of significant interstitial lung disease." Dr. Spagnolo stated that the miner "probably has mild airflow obstruction caused by his long use of cigarettes." In particular, Dr. Spagnolo pointed to the normal FVC values on multiple occasions, normal total lung capacity in 1989, and normal blood gas values in 1992 and 1993. Dr. Spagnolo concluded that the miner was not totally disabled from a respiratory standpoint, but that the miner's coronary artery disease could render him totally disabled.

On June 24, 2002, Dr. Spagnolo issued a second report after review of certain medical records. *Ex.* 5. He concluded that the miner did not suffer from coal workers' pneumoconiosis nor did he suffer from a totally disabling respiratory impairment. Dr. Spagnolo reported 28 years, of coal mine employment, where the miner last worked from 1981 to 1989 as a dragline operator. He also reported a 23 year history of smoking one to four packs of cigarettes per day, quitting in 1962 or 1963. A medical history of the miner revealed diabetes, coronary artery disease, arthritis, hypertension, obesity, vascular disease, and chronic low back pain. Dr. Spagnolo stated that he would give "greater weight" to the "thoughtful, well-reasoned (chest x-ray) interpretations" of Drs. Wheeler, Wiot, Spitz, and Scott, who did not find the presence of coal workers' pneumoconiosis, because of their "extensive experience." Moreover, Dr. Spagnolo noted that Drs. Naeye, Caffrey, and Hutchins found generally insufficient tissue evidence of pneumoconiosis on autopsy. Dr. Spagnolo stated that the miner's mild respiratory impairment was not due to coal workers' pneumoconiosis, although he provided no other cause for the impairment. He further concluded that pneumoconiosis did not contribute to the miner's total disability or death.

Dr. Spagnolo was deposed on October 16, 2003. *Ex.* 11. He is a Professor of Medicine at the George Washington University Medical Center. *Ex.* 1. He serves as a lecturer on pulmonary and critical care medicine and is board-certified in internal medicine with a sub-specialty in pulmonary disease. During his deposition, Dr. Spagnolo stated that, although the miner stopped smoking in the early 1960s, his smoking-induced lung disease "would continue on the rest of his life." *Ex.* 11 at 14. Dr. Spagnolo noted the following:

> The damage . . . that is incurred by the cigarette smoking in terms of the primary lung damage to the interstitium of the lung and alveolar part of the lung is a permanent process, so that doesn't go away after you stop smoking.

Dr. Spagnolo stated that, prior to the miner's death, he suffered from "severe probable end-stage heart disease from coronary artery disease" and the miner "was having a great deal of difficulty" prior to his death. *Ex.* 11 at 20. He noted that the January 1986 pulmonary function testing was within normal limits and revealed no significant impairment. *Ex.* 11 at 24, 26. Dr. Spagnolo found that the August 1996 pulmonary function study showed "great variability," which was inconsistent with a "static disease" like coal workers' pneumoconiosis. *Ex.* 11 at 26. He stated that the variability in lung function was due to the miner's long-time smoking history and his cardiovascular disease.

Dr. Spagnolo concluded that the miner did not suffer from coal workers' pneumoconiosis or, if it was present, "it had to have been in such an extremely limited or minor condition as to not have any impact on this man's pulmonary function or lung condition." *Ex.* 11 at 31.

With regard to findings of emphysema on autopsy, Dr. Spagnolo stated that coal workers' pneumoconiosis can cause focal emphysema. *Ex.* 11 at 79. However, centrilobular emphysema stems from tobacco abuse. *Ex.* 11 at 82. Dr. Spagnolo noted that centrilobular emphysema in cigarette smokers is always accompanied by bronchiolitis, but Dr. Naeye found no bronchiolitis upon his examination of the miner's lungs. *Ex.* 11 at 84. On the other hand, bronchiolitis is absent from persons suffering from focal dust emphysema. *Ex.* 11 at 90. Finally, Dr. Spagnolo stated that coal workers' pneumoconiosis may cause chronic bronchitis in some individuals. *Ex.* 11 at 96.

DR. RICHARD L. NAEYE

Dr. Richard Naeye reviewed the autopsy report, a copy of the death certificate, 53 slides, and certain other medical records and he issued an opinion on June 19, 1999. *Dx.* 71. Dr. Naeye noted 29 years of working at a strip mine, where Mr. Schutt retired in 1989. He also noted a 21 year history of smoking one to four packs of cigarettes per day, quitting in 1963. Many chest x-ray interpretations were negative for the presence of pneumoconiosis and the miner suffered a myocardial infarction in January 1997 and died of cardiac arrest on June 2, 1997.

Dr. Naeye reported that Dr. Dikman noted the presence of a 1.3 centimeter "anthrasilicotic macule" in the miner's left lower lobe. He found minimal fibrosis, but noted severe, diffuse pulmonary edema. On microscopic examination, Dr. Naeye found no more black pigment than is found in most non-miner's lungs. With regard to the anthrasilicotic macule, Dr. Naeye noted that it was 1.0 in diameter at its greatest dimension and "[i]t has only a tiny amount of black pigment in it so it cannot be described as anthracotic." He found that the miner's centrilobular emphysema varied from mild to moderately severe, which "correlates with the normal 1995 results of pulmonary function and arterial blood gas analysis." Dr. Naeye stated that old silicotic lesions were comprised of irregular layers of hyalinized collagen and mixed birefringent crystals of all sizes. He further noted that the "hyalinized collagen is also not in the irregular layered configuration characteristic of silicosis."

Dr. Naeye stated that the ratio of mucous to serous glands in the walls of the bronchi was about 3:1, as opposed to the normal ratio of 1:1. This constituted evidence of chronic bronchitis. However, he found that chronic bronchiolitis was absent and there was no evidence of asbestosis. In sum, Dr. Naeye concluded that the miner did not suffer from simple or complicated pneumoconiosis and that fluctuations in his pulmonary function testing were due to fluctuations in his chronic bronchitis. He stated that the miner's "fatal acute pulmonary edema was the consequence of very sudden onset acute cardiac failure." Because coal workers' pneumoconiosis was absent, Dr. Naeye found that the disease could not have contributed to the miner's total disability or death.

Dr. Naeye was deposed on October 14, 2003. *Ex.* 10. Dr. Naeye is board-certified in anatomic and clinical pathology. *Ex.* 10 at 4. He helped to found the medical department at

Pennsylvania State University College of Medicine and University Hospital and he served as Chair of the department for 30 years. *Ex*. 10 at 4. Dr. Naeye currently serves as a professor and he teaches students about pulmonary disease. *Ex.* 10 at 5. In 1979, he published an article on the "Pathology Standards for the Diagnosis of Coal Workers' Pneumoconiosis." *Ex*. 10 at 8. He asserts that, as far as diagnosis of the disease is concerned, his standards are still valid today. *Ex.* 10 at 8. Over the years, Dr. Naeye has reviewed 2,000 or 3,000 cases for the Department of Labor to determine the presence of coal workers' pneumoconiosis, but he quit two to three years ago because the Department could not get all the materials together for a single review and he was required to conduct his reviews piecemeal. *Ex.* 10 at 8-9.

Initially, Dr. Naeye noted that Mr. Schutt spent 29 years as a strip miner and that this was a "very important issue." *Ex.* 10 at 11. In this vein, Dr. Naeye noted:

> The exposures are complex underground and you have to know a lot about the exact job they were doing and how long they were doing it and the location of the mine. The problems with strip mining are a little different.

Ex. 10 at 11.

With regard to the autopsy of the lungs, Dr. Naeye stated that he did not conduct a gross examination of the lung tissue. *Ex.* 10 at 14. He noted that this may or may not be disadvantageous but that "[i]n this particular case, the persons who looked at the gross were people with experience." *Ex.* 10 at 14. Dr. Naeye was referring to Dr. Kleinerman, even though Dr. Dikman also conducted a gross examination of the lung tissue. *Ex.* 10 at 14-15.

Dr. Naeye stated that any findings with regard to asbestos were irrelevant as he had no knowledge that the miner was exposed to asbestos. *Ex.* 10 at 16. Dr. Naeye reported that coal workers' pneumoconiosis was not present on the autopsy slides and the "microscopic examination confirm(ed) massive severe acute pulmonary edema due to heart failure or pneumonia or both. *Ex.* 10 at 20-21.

Dr. Naeye observed a one centimeter lesion in the lower lung and stated the following with regard to its composition:

> It only had a tiny amount of black pigment in it so it could not be described as anthracotic. That's important because the black pigment itself is carbon in an amorphous form that is nontoxic and it remains in the tissues forever. It will decrease slowly after retirement from mining coal, but I can't remember ever seeing a coal miner that didn't have black pigment in his lungs at the time of death.

Ex. 10 at 21. Dr. Naeye stated that the lesion was one centimeter at its "greatest dimension" and it was not silicotic in origin. *Ex.* 10 at 21. He observed no tiny birefringent crystals or free silica in the lesion and, because "it wasn't layered," the lesion must have been a "healed lesion of something" but it was not silicotic in origin. *Ex.* 10 at 22.

Dr. Naeye found that mild to moderately severe centrilobular emphysema was present, but there was no evidence of bronchiolitis. *Ex.* 10 at 23. He concluded that "smoking has about three times the role of coal mine dust exposure in terms of causing centrilobular emphysema." *Ex.* 10 at 23. Dr. Naeye opined that the emphysema would not have prevented Mr. Schutt from engaging in his last coal mine employment. *Ex.* 10 at 23. He also noted that the miner suffered from mild to moderately severe chronic bronchitis from cigarette smoking. *Ex.* 10 at 24.

Dr. Naeye stated that the cause of the miner's death was "very sudden onset of severe cardiac failure" that caused pulmonary edema. *Ex.* 10 at 25. Dr. Naeye did not take issue with Dr. Kleinerman's finding of a few macules of coal workers' pneumoconiosis but, "[b]ecause there was so little black pigment in this man's lung, there weren't any significant lesions." *Ex.* 10 at 26. Dr. Naeye concluded that the miner's chronic lung disease did not affect the course of his death; rather, he suffered a "cardiac death." *Ex.* 10 at 26.

On cross-examination, Dr. Naeye noted the Dr. Kleinerman found sufficient macules with fibrosis to diagnose coal workers' pneumoconiosis, but Dr. Naeye did not agree with that assessment. *Ex.* 10 at 37. Dr. Naeye also never mentions the miner's lymph nodes, whereas Dr. Dikman did make mention of the lymph nodes. *Ex.* 10 at 47. Dr. Naeye acknowledged that he "never engaged in any energy dispersive spectroscopy or electron defraction analysis relative to the parenchyma or the lung tissue from the parenchymal lung tissue." *Ex.* 10 at 53. Moreover, he did not conduct a tissue digestion or electron microscopy. *Ex.* 10 at 54. Dr. Naeye stated that he looked for free silica particles in all lung samples containing black pigment, but he did not find "enough free silica particles that would cause fibrosis or any disease process." *Ex.* 10 at 55. He stated that he did not mention the presence of silica particles in his report because he must observe a certain number of such particles before finding them clinically significant. *Ex.* 10 at 55.

DR. BRIAN P. DOLAN

Dr. Brian P. Dolan is board-certified in internal medicine, preventative medicine, and occupational medicine. He has a Master's degree in Public Health and has a private practice.

Dr. Dolan reviewed a large number of medical records and issued his first report on January 9, 1997. *Dx.* 58; *Claimant's Exhibit (Cx.)* 15A (attached to his September 2003 deposition). In this report, Dr. Dolan noted the miner's work and smoking histories and treatment notes to find that the miner was totally disabled from coal workers' pneumoconiosis. He noted that the treating physicians recorded severe respiratory symptoms and that the miner used oxygen by nasal cannula at home. Dr. Dolan opined that the miner suffered from chronic obstructive pulmonary disease arising from coal mine employment and cigarette smoking. Dr. Dolan stated the following:

> His total disability is based on clinical and laboratory data. His spirometry and arterial blood gas values as of February 7, 1995 are slightly above the values set for total disability. The chest x-ray reading of Dr. Kimmel on the same date suggests cor pulmonale but does not prove it. On the other hand, his treating doctors have recorded severe respiratory symptoms, such as inability to mow even

> with a power mower (1991), having to stop to breathe three times walking from the parking lot to the clinic (1992), dyspnea on minimal exertion (1993), gradually worsening dyspnea on exertion (1995). He currently uses oxygen by nasal cannula. The latest pulmonary exercise test I have reviewed, dated 2/10/95, showed severe exercise limitation. He could only exercise at low exertion level for 6 minutes, dropping his oxygen saturation from 92 to 85. At an oxygen saturation of 90% (equaling a PO2 of approximately 60), most people will have the sensation of being short of breath. After his very modest exertion, with an oxygen saturation of 85% (PO2 of 53.6), he probably felt very uncomfortably short of breath. According to the physician's comments, his condition was worsening with time. On the basis of symptoms noted by his physician and corroborated by the exercise testing, Mr. Schutt cannot perform any reasonable work and presently seems to require supplemental O2 just for activities of daily living.

Dr. Dolan reviewed certain medical records and issued a report dated September 20, 2003. *Cx.* 1A, 15A. He noted that, according to the death certificate, the miner died of cardio-respiratory arrest on June 2, 1997. Dr. Dolan reported a 30 year history of coal mine employment and a 50 pack year of smoking cigarettes, where the miner stopped in 1962. Upon review of the miner's treatment records, Dr. Dolan noted that the miner "had symptomatic lung disease which began in 1975 with subsequent and progressive deterioration." A 1993 evaluation revealed evidence of an obstructive and restrictive lung disease along with decreased diffusing capacity. At that point, however, there was no medical evidence of heart disease. Indeed, the medical records supported a finding that the miner did not suffer from disabling heart disease at that time. In 1996, the miner had developed pulmonary hypertension and cor pulmonale as well as probable coronary artery disease. He was place on home oxygen in August of 1996. By 1997, the miner's breathing worsened and he had developed coronary artery disease. Accordingly, Dr. Dolan stated that the miner suffered from a disabling respiratory disease arising from his coal mine employment, which existed prior to the development of his cardiac condition.

Dr. Dolan was deposed on September 22, 2003. *Cx.* 15A. He stated that, in 1978 and 1979, he "was the physician for the area around Rough Rock in the Navajo Reservation in Arizona" and "a large number of the people who lived in that community worked in the Peabody Coal mines, which were in the adjacent Black Mesa." *Cx.* 15A at 9. Dr. Dolan has also treated people with asbestosis. *Cx.* 15A at 10-11.

Dr. Dolan stated that he reviewed the autopsy report and certain other medical records of the miner and that, based on the miner's hospitalization and treatment records, it was "more likely than not" that the miner suffered from cor pulmonale. *Cx.* 15A at 23. Moreover, Dr. Dolan noted that the lung autopsy rendered his 1997 diagnosis of asbestosis untenable. *Cx.* 15A at 23, 44-45. He reported that Dr. Dikman diagnosed the presence of carbon pigments and silicates with associated fibrosis, which qualifies as a diagnosis of coal workers' pneumoconiosis. *Cx.* 15A at 32, 44-45. Dr. Dolan noted that Dr. Kleinerman agreed that the miner suffered from a very mild degree of simple coal workers' pneumoconiosis. *Cx.* 15A at 41. He found that all of the pathologists agreed that "pulmonary emphysema, interstitial fibrosis, and pleural plaquing" was present in the miner's lungs. *Cx.* 15A at 54. However, unlike any other

pathologist analyzing the miner's lung tissue or looking at his slides, Dr. Dolan noted that Dr. Dikman performed "digestion techniques" and, based on this procedure, he found "significant amounts of silica in the lung and lymph node tissue." *Cx.* 15A at 54-55. Dr. Dolan described the digestion process:

> [L]ung tissue and lymph node tissue is placed in a solution of a chemical that dissolves away the soft tissue and leaves only the mineral content of the lung and lymph node. In this case, the mineral content was silicates and talc.

Cx. 15A at 71.

Dr. Dolan also observed that Dr. Dikman found "talc" in the lungs and that, from his reading of literature, talc is often found in the lungs of miners and he "assume(d) that it's a contaminant or other dust that coal miners routinely encounter in their work." *Cx.* 15A at 70.

He reported a 50 pack year history of smoking cigarettes, stopping in 1962, and a 30 year coal mine employment history, where the miner worked from 1959 to 1989. *Cx.* 15A at 43. Dr. Dolan also found, in Dr. Graham's September 16, 1999 report, that the miner has a history of working in a coal pit for six years. *Cx.* 15A at 34-35. The miner stopped smoking in 1962, "many years before he developed any respiratory symptoms." *Cx.* 15A at 43. According to Dr. Dolan's review of the medical records, the miner's shortness of breath started in 1975 and worsened with time. *Cx.* 15A at 43. Further, Dr. Stoy's 1993 examination revealed obstructive and restrictive pulmonary disease on pulmonary function testing, decreased diffusion capacity, and very poor exercise tolerance. A chest x-ray revealed a normal heart, no evidence of congestive heart failure, and pulmonary fibrosis in the lungs. *Cx.* 15A at 43.

By 1996 and 1997, Dr. Dolan noted that the miner was hospitalized for coronary artery disease, which was "successfully treated by angioplasty." *Cx.* 15A at 44. During a 1996 hospitalization, a thallium test demonstrated right ventricular decompensation. *Cx.* 15A at 44. In May 1997, the miner was hospitalized for congestive heart failure and, by June 1997, he had developed back pain and "had a respiratory arrest from which he could not successfully be resuscitated." *Cx.* 15A at 44.

Dr. Dolan stated that he disagreed with Dr. Lange's conclusion as to the cause of death on the miner's death certificate and noted that, in his emergency room report, Dr. Lange was uncertain as to whether the miner's death was cardiac or pulmonary and "in fact, the history he reported seemed to emphasize his severe pulmonary disease." *Cx.* 15A at 50. Dr. Dolan further stated:

So I was at a loss to explain why (Dr. Lange) wrote on the death certificate that the cause of death was cardiac arrest with arrhythmia due to arteriosclerotic cardiovascular disease when there's no indication that he had any further information which would sway him more toward cardiac than pulmonary.

Cx. 15A at 50.

Dr. Dolan concluded that he agreed with Dr. Stoy that the miner suffered from a disabling lung condition, not a disabling cardiac condition. *Cx.* 15A at 50. Dr. Dolan stated the following:

> And the only question is: To what degree was his lung (disease) disabling? And to determine this, I feel most comfortable looking at the pulmonary exercise data in 1992.

[I]t's my understanding that the level of maximum oxygen uptake was 12, which would place him in Category 4 (of the American Medical Association Guidelines), the most severe, the category which would mean he was totally disabled at that time.

Another pulmonary exercise test in 1995 was 17.5, which would put him in the moderately disabled. So I'm not sure how to explain this discrepancy. So he was either totally disabled in 1992 if you believe the 1992 data, or was not totally disabled at least by the criteria of exercise testing until some time in 1995 or 1996. By 1996 his blood oxygen had dropped below the level of 60.

Cx. 15A at 50-51. Dr. Dolan stated that, with an oxygen level of 60, the average person would feel short of breath even at rest. *Cx.* 15A at 51. He noted that the miner was on supplemental oxygen at home in August 1996. *Cx.* 15A at 52.

With regard to the 1995 study revealing an oxygen uptake of 17.5, Dr. Dolan stated that "this exercise level shows that (the miner) wasn't even able to reach his maximum predicted heart rate because of shortness of breath." *Cx.* 15A at 77-78. Dr. Dolan further stated the following:

> (The miner) didn't have congestive heart failure by that time. And actually the report says that he had a normal cardiovascular response to exercise, meaning no deconditioning and that he had abnormal pulmonary response to exercise which the pulmonologist said was due to a mix-up of structural and restrictive defects . . ..
>
> So I think this test, if anything, shows that his heart rate, that his heart responses were normal, that he had no obvious deconditioning, although you would expect that he might have some. And that the disability and limitation was due to ventilatory or pulmonary limitation.

Cx. 15A at 77-78. He further noted that the use of bronchodilators on pulmonary function testing "did not appear helpful." *Cx.* 15A at 94.

Dr. Dolan concluded that the miner suffered from coal workers' pneumoconiosis, which progressively worsened during his lifetime, and contributed to his total disability during his lifetime and hastened his death. *Cx.* 15A at 96, 99, 101.

DR. W.K.C. MORGAN

Dr. W.K.C. Morgan issued his first report on June 12, 1995, after reviewing certain medical records. *Dx.* 57. Based on the medical data available to him, Dr. Morgan concluded that the miner suffered from a mild airways obstruction. He noted that, "[e]arlier on there was no evidence of restrictive impairment, however, there has been a gradual decline in both the FVC and FEV1." Dr. Morgan stated that it was not "marked," but he believed that the miner had a "minimal restrictive impairment and this has developed as he has put on weight." He also noted that the lower FVC value during 1993 testing could have been the consequence of interstitial fibrosis and a collapsed lower lung, or it could be due to his being overweight. Dr. Morgan noted that a majority of x-ray interpretations, particularly the readings of Dr. Wiot and his colleagues, do not support a finding of coal workers' pneumoconiosis. Dr. Morgan concluded that the miner's respiratory impairment was not totally disabling although he has other factors, such as obesity and being 70 years of age, that could contribute to his disability. Dr. Morgan opined that the miner's respiratory impairment was unrelated to coal dust exposure and that, even if the miner suffered from coal workers' pneumoconiosis, any resulting impairment would be entirely due to his smoking history as the miner was a heavy smoker.

Dr. Morgan reviewed additional medical records and issued a second report on July 15, 2002. *Ex.* 8. He is an honorary Fellow on the Faculty of Occupational Medicine at the Royal College of Physicians in London and serves as a Professor of Medicine at the University of West Ontario. Dr. Morgan is also a B-reader.

Dr. Morgan noted that Mr. Schutt worked at the strip mines for 28 years until 1989. From 1971 through 1989, he was a dragline operator and from 1971 to 1976 he was exposed to asbestos from maintaining friction brakes. Dr. Morgan also reported a 23 year history of smoking one to four packs of cigarettes per day, where the miner quit in 1962 or 1963.

Pulmonary function testing revealed a decreased FEV1 over the years and Dr. Morgan stated that "[t]his would suggest that Mr. Schutt had mild airways obstruction which must be attributed to the fact that when has was young he was a heavy cigarette smoker." The miner's reduced diffusing capacity "can be attributed to his former habit of cigarette smoking which caused his emphysema along with the fact that his cardiac output was reduced." Dr. Morgan further found that a majority of B-readers did not find the presence of coal workers' pneumoconiosis by chest x-ray.

Dr. Morgan concluded that it "is most unusual for strip miners to develop coal workers' pneumoconiosis" and that Mr. Schutt had no evidence of the disease. Rather, he suffered from a "very mild obstruction and later on developed some restrictive impairment when his cardiac disease became worse." Dr. Morgan speculated that the miner's obesity "may have contributed to his restriction."

Dr. Morgan found that the miner was totally disabled from cardiac problems unrelated to coal mine employment. He stated that there was an insignificant presence of macules on autopsy to qualify for the diagnosis of coal workers' pneumoconiosis and that the macules were "perfectly harmless and (did) not produce any impairment."

DR. GREGORY FINO

Dr. Gregory Fino reviewed certain medical records and issued his initial report on November 15, 1993. *Dx.* 27. He noted 28 years of coal mine employment where the miner retired in 1989 as a dragline operator. He also reported a 21 year history of smoking one to four packs of cigarettes per day, stopping in 1963.

Based on his review of medical records, Dr. Fino concluded that the miner did not suffer from coal workers' pneumoconiosis. He noted that a majority of chest x-ray readings, including five studies reviewed by him, did not indicate the presence of pneumoconiosis:

> What I noted on the chest x-ray, and what many others noted, was a generalized increase in irregular or interstitial markings in the lower lung zones. This is not consistent with coal workers' pneumoconiosis.

Additionally, Dr. Fino noted that pulmonary function testing revealed a pure obstructive ventilatory abnormality without restriction:

> This type of finding is not consistent with a coal dust related condition but is consistent with conditions such as cigarette smoking, pulmonary emphysema, non-occupational chronic bronchitis, and asthma. This type of pattern is consistent with a pure obstructive ventilatory abnormality as would be seen in asthma or in conditions related to cigarette smoking.

He noted that the miner's diffusing capacity values were normal, which ruled out the presence of a clinically significant pulmonary fibrosis and "[p]neumoconiosis is, of course, an example of a pulmonary fibrosis."

Dr. Fino also noted that the miner had a "marked elevation in lung volumes" and stated the following:

> There is stale air trapped in his lungs due to his obstructive lung disease. This is a typical pattern that we see in individuals who have obstructive lung diseases such as emphysema, or asthma, or chronic obstructive bronchitis, or any combination of three. This is not a pattern consistent with the contraction of lung tissue due to fibrosis as would be expected in simple coal workers' pneumoconiosis.

Dr. Fino opined:

> One would expect in a coal dust related impairment a reduction in the lung volumes indicating fibrosis along with a reduction in diffusion. In this case, however, we have markedly elevated lung volumes consistent with air trapping. Also, there is a normal diffusing capacity.

In sum, Dr. Fino found insufficient objective medical evidence to diagnose simple coal workers' pneumoconiosis. He concluded that the miner did not suffer from an occupationally-acquired pulmonary condition and that he was not totally disabled "unless his last classified job included continuous heavy manual labor."

Dr. Fino reviewed certain medical records and issued a second report on June 12, 1995. *Dx.* 57. Based on chest x-ray interpretations and pulmonary function testing, Dr. Fino stated the following:

> I believe that the interstitial changes in the lower lung zones are either fibrosing alveolitis which is a form of diffuse interstitial fibrosis unrelated to the inhalation of coal mine dust, or bronchiectasis, which is an abnormal dilation of the breathing tubes associated with scarring and fibrosis which has nothing to do with the inhalation of coal mine dust.

Blood gas testing "clearly show an impairment in oxygen transfer consistent with an interstitial type of abnormality in the lungs." Dr. Fino opined that coal workers' pneumoconiosis occurs in the upper, not lower, lungs. He found that the miner had a significant oxygen transfer abnormality since 1989 such that he could not perform heavy labor, but the miner could perform duties as described by Dr. Stoy.

Dr. Fino again reviewed certain medical records and issued a report on June 14, 2002. *Ex.* 7. He is board-certified in internal medicine and pulmonary disease and he is a B-reader. Dr. Fino stated that he would assume that coal workers' pneumoconiosis was present based on the autopsy evidence. However, he concluded that there was an insufficient level of coal workers' pneumoconiosis on pathology to account for the oxygen transfer abnormalities seen on the miner's testing. Dr. Fino stated the following:

> [T]aking all of the pathologists' evaluations together, there certainly was not enough intrinsic lung disease to account for an oxygen transfer abnormality. However, there was a sufficient smoking history to account for that.

Dr. Fino opined that the miner died from cardiac arrest unrelated to coal dust exposure. Rather, he stated that the miner had an acute condition, noting that the miner's 1997 blood gas study values while terminally ill were not valid and they reflected his cardiac distress as opposed to a chronic respiratory impairment. Dr. Fino concluded that the miner suffered from a mild respiratory impairment due to smoking and that he was totally disabled from his last job, but not because of any coal dust induced disease.

DR. JAMES CASTLE

Dr. James Castle reviewed certain medical records and issued his initial report on June 12, 1995. *Ex.* 4 (attached to his January 1997 deposition). At the time, Dr. Castle concluded that the miner did not suffer from coal workers' pneumoconiosis. He noted a 28 year history of working at the mines and that the miner's "history indicates that this included a limited exposure to significant amounts of coal dust. Dr. Castle stated that it was "not clear that he actually was

exposed to sufficient quantities of coal dust during his employment history to have developed coal workers' pneumoconiosis." However, Dr. Castle "assumed" significant exposure.

Dr. Castle noted that physical examinations on a number of occasions yielded "evidence of rales, crackles, or crepitations particularly involving the lower lung zones." He further found that most B-readers did not find pneumoconiosis present on chest x-ray. He stated that many pulmonary function studies did not have tracings attached, which precluded a determination of their validity. One study by Dr. Fino "showed a totally obstructive ventilatory pattern with no evidence of restriction and (Dr. Fino) felt this was evidence of only an obstructive airways disease and did not indicate any evidence of pneumoconiosis." Dr. Castle stated that the December 1993 pulmonary function study showed a precipitous drop in values from the November 30, 1993 study (only two weeks earlier), which demonstrated an acute (not chronic) disease process. Dr. Castle further found that blood gas study values were normal or showed mild hypoxemia "with an essentially normal response to exercise."

Dr. Castle concluded that the miner had a mild degree of pulmonary impairment due to a mild obstructive ventilatory defect demonstrated on valid pulmonary function testing, but this was related to his smoking history since there was no radiographic or pulmonary function study evidence of coal workers' pneumoconiosis. The miner did not suffer from any restrictive impairment, as would be seen with an interstitial pulmonary process such as asbestosis or coal workers' pneumoconiosis. As a result, Dr. Castle concluded that the miner was not totally disabled from a respiratory standpoint, but that he could be disabled from other factors such as his age or diabetes.

At the time of his January 21, 1997 deposition, Dr. Castle opined that the miner did not suffer from coal workers' pneumoconiosis. *Ex.* 4 at 9. In support of this conclusion, he stated that the miner did not consistently have rales, crackles, or crepitations, there were no small rounded opacities seen on the chest x-rays, there was no restrictive ventilatory impairment (and coal workers' pneumoconiosis would cause an irreversible obstructive and restrictive impairment), and there were no blood gas findings to corroborate the diagnosis of coal workers' pneumoconiosis. *Ex.* 4 at 10. Dr. Castle stated that the miner had a mild level of impairment that was related to a mild obstructive ventilatory defect. *Ex.* 4 at 11.

Dr. Castle stated the diffusion capacity is designed to measure the "lungs' ability to take up carbon monoxide." *Ex.* 4 at 13. He stated the following:

> When people have either a fibrotic process in the lungs, such as coal workers' pneumoconiosis or other forms of fibrosis, or if they have a significantly destructive process in the lung, such as emphysema, they will have a reduction in the diffusion capacity for carbon monoxide.

In this case, Dr. Castle noted that the miner had a reduced diffusing capacity, but "when it was corrected for volume, it was essentially normal, or at the lower limit of normal." *Ex.* 4 at 14. Dr. Castle concluded that the miner was not totally disabled from a respiratory standpoint, since the work of a dragline operator required "minimal actual work." *Ex.* 4 at 15. However, the miner's respiratory impairment was related to tobacco smoking and not coal dust exposure. *Ex.* 4 at 15.

Dr. Castle opined that the miner's 20 year history of smoking more than one pack of cigarettes per day caused him to develop "chronic obstructive pulmonary disease, which is manifested by the mild degree of obstruction on his pulmonary function tests, that clearly appears to be chronic bronchitis in nature." *Ex.* 4 at 16. Dr. Castle stated that, if the miner suffered from coal workers' pneumoconiosis, there should be x-ray evidence of the disease as well as consistent findings of crackles, a restrictive ventilatory process, clubbing, and a reduced diffusing capacity. *Ex.* 4 at 17.

Dr. Castle opined that the miner did not have legal pneumoconiosis. *Ex.* 4 at 23. He noted that coal workers' pneumoconiosis "may be associated with an mild irreversible obstructive component to the overall situation associated with some restrictive lung disease." *Ex.* 4 at 23. He concluded that the obstructive findings in this case were not related to coal workers' pneumoconiosis because the miner did not "have any other findings to indicate the presence of that disease, other than historical data." *Ex.* 4 at 24.

On cross-examination, Dr. Castle stated that he was not board-certified in occupational disease and he had not published any articles on coal workers' pneumoconiosis or asbestosis. *Ex.* 4 at 28-29. He acknowledged that pathological findings are more probative than chest x-ray interpretations and that the miner did have some small airways disease, but it was an insignificant part of the obstructive disease which, in turn, was due to tobacco smoking. *Ex.* 4 at 56.

Dr. Castle reviewed certain medical records and issued a supplemental report on June 13, 2002. *Ex.* 6. He is board-certified in internal medicine, with a subspecialty in pulmonary diseases, and he is a B-reader. *Ex.* 4 at 4. Dr. Castle is also on staff at the Carolion Roanoke Memorial Hospital and Carilion Roanoke Community Hospital and has an active pulmonary practice, where he sees 14 to 15 patients each day at the hospital and 14 to 18 patients per day at the office. *Ex.* 4 at 5-6. Dr. Castle is also a clinical professor at the University of Virginia School of Medicine. *Ex.* 4 at 7.

In his report, Dr. Castle concluded that the miner "possibly did have pathologic evidence of coal workers' pneumoconiosis." He explained that "[t]his process was not found clinically, but several pathologists felt that there were very minimal changes which would constitute adequate pathologic evidence to render a diagnosis of coal workers' pneumoconiosis." He reported 28 years of coal mine employment, where the miner last worked in 1989, and 20 years of smoking one to four packs of cigarettes per day. Dr. Castle noted that rales, crackles, and crepitations, which would indicate the presence of a chronic interstitial pulmonary disease such as asbestos or coal workers' pneumoconiosis, were "not there on a consistent basis." He further stated that a majority of B-readers did not find the presence of the disease by chest x-ray. Valid pulmonary function studies showed evidence of mild to moderate pure airway obstruction, which was related to the miner's long, extensive tobacco smoking habit. Dr. Castle concluded that the miner probably did not retain the respiratory capacity to return to his previous coal mine employment, but this impairment was not related to coal dust exposure. With regard to pathological findings of the disease, Dr. Castle noted that "this process was so minimal as to have not caused him any impairment during life and it could not have caused, contributed to, or hastened his death in any way." He stated that his opinion was "based upon the sparsity of

findings in the pathologic specimens and the lack of physiologic impairment associated with coal workers' pneumoconiosis."

DR. LAWRENCE REPSHER

By report dated December 3, 1996, Dr. Lawrence Repsher concluded that the miner did not suffer from coal workers' pneumoconiosis. *Dx.* 57. He reported 28 years of coal mine employment and a 23 year smoking history of up to four packs of cigarettes per day. Dr. Repsher noted that all of the pulmonary function studies "document moderate chronic obstructive pulmonary disease without significant immediate bronchodilator response, quite consistent with his history of intense cigarette smoking." Dr. Repsher opined that the miner's low diffusing capacity "suggests significant underlying emphysema." He explained the following:

> It should be pointed out that the lung volumes, as confirmed by all of the chest x-rays, document pure obstructive disease without any significant restrictive component. Coal workers' pneumoconiosis, where clincially significant, is primarily a restrictive disease that may have some obstructive features. In summary, the pulmonary function abnormalities are most consistent with chronic obstructive pulmonary disease and emphysema and inconsistent with a restrictive disease, such as coal workers' pneumoconiosis.

Dr. Repsher concluded that the miner did not suffer from coal workers' pneumoconiosis; rather, he had moderately severe chronic obstructive pulmonary disease and emphysema that was "overwhelmingly most likely due to Mr. Schutt's prior history of heavy cigarette smoking." He noted that chest x-rays did not reveal the presence of the disease and blood gas study values improved over time, which was inconsistent with a finding of coal workers' pneumoconiosis. Moreover, pulmonary function testing revealed a pure obstruction without any evidence of a restrictive component as would be seen with coal workers' pneumoconiosis and these abnormalities were characteristic of cigarette smokers like the miner who have developed chronic obstructive pulmonary disease and emphysema.

Dr. Repsher reviewed certain medical records and issued a second report on September 18, 2001. *Ex.* 3. He is the Medical Director of the Occupational and Environmental Lung Disease Program at the Lutheran Medical Center. Dr. Repsher is a NIOSH B-reader and is board-certified in internal medicine with subspecialties in pulmonary disease and critical care.

Dr. Repsher noted a 28 year history of coal mine employment as well as a 23 year history of smoking one to four packs of cigarettes per day, quitting in 1962 or 1963. In his medical record review, Dr. Repsher noted that most of the B-readers interpreted the chest x-rays as negative for the presence of coal workers' pneumoconiosis. He noted that, prior to developing congestive heart failure, the miner "exhibited mild to moderate pure obstructive disease with no element of restrictive disease" and no reversibility. He concluded that this was not typical of clinically significant coal workers' pnuemoconiosis.

Dr. Repsher further found that the miner's diffusing capacity was moderately to moderately severely impaired, which was "consistent with the significant centrilobular emphysema noted at autopsy." He diagnosed the miner as suffering from mild to moderate chronic obstructive pulmonary disease with centrilobular emphysema, severe coronary artery disease, vessel disease, diabetes mellitus, obesity, and hypertension.

Dr. Repsher concluded that the miner did not suffer from coal workers' pneumoconiosis during his lifetime and his death was not hastened by the disease. He based this conclusion on the following: (1) no chest x-ray evidence of the disease; (2) pulmonary function testing revealed a purely obstructive disease and "coal workers' pneumoconiosis, when clinically significant, is primarily a restrictive disease that may have some obstructive features"; and (3) there was no blood gas study evidence of coal workers' pneumoconiosis as a miner with the disease generally has a low carbon dioxide level, but Mr. Schutt had a tendency to have a high carbon dioxide level. Dr. Repsher concluded that the miner's problems were due to lifestyle and heredity. His chronic obstructive pulmonary disease was due to his cigarette smoking and he suffered from coronary artery disease.

Dr. Repsher also testified at the October 2003 hearing. *Tr.* at 74. Based on his review of the medical records, Dr. Repsher concluded that the miner had "mild – possibly moderate – it's hard to tell because there was a great deal of variation in his effort . . ., but he at least had some COPD from his cigarette smoking." *Tr.* at 81. Dr. Repsher concluded that smoking alone caused the miner's respiratory impairment:

> [O]ne, he had a very significant history of cigarette smoking. Two, he had only minimal histologic evidence of coal workers' pneumoconiosis. And, three, the COPD that is associated with inhalation of coal mine dust on the average is so small that it cannot be detectable in the measurements of a single individual, the reason being that the change in the FEV sub 1 is much smaller – only a small fraction of the anticipated test-to-test and day-to-day variation of just repeating the spirometry tests.

Tr. at 82. Focusing on the pulmonary function testing of record, Dr. Repsher concluded that the miner suffered from a mild respiratory impairment. *Tr.* at 83. He stated that the medical data supported a finding that the miner developed his first evidence of congestive heart failure in 1995 or 1996. *Tr.* at 85.

Dr. Repsher noted that the miner's exercise testing was increasingly abnormal due to his "serious organic heart disease." *Tr.* at 87. Dr. Repsher opined that the miner used supplemental oxygen because his "lung (was) full of fluid from congestive heart failure . . .." *Tr.* at 89.

DR. MICHAEL GRAHAM

Dr. Michael Graham is a professor of pathology at the St. Louis University School of Medicine and he reviewed certain medical records and issued a report on September 16, 1999. *Cx.* 15A (attached as exhibit to Dr. Dolan's September 2003 deposition). He works in the Department of Pathology, Division of Forensic and Environmental Pathology. Dr. Graham

reported a 21 year history of smoking two to three packs of cigarettes per day, where the miner quit in 1962. He also reported 30 years of coal mine employment and that the miner last worked as a dragline operator, where he was exposed to asbestos while working on brakes and brake linings.

Dr. Graham reviewed the autopsy report of Dr. Dikman, analyzed the formalin-fixed right and left lungs, and considered certain medical records in his report. Upon examination, Dr. Graham noted that the miner's lungs contained "patchy interstitial fibrosis," "mild fibrosis of an occasional respiratory bronchiolar wall," a "moderate amount of punctate and needle-like crystalline particles," a "pleural silicotic nodule," and emphysema. Dr. Graham noted that "[t]here is an occasional interstitial deposit of carbonaceous particulate material associated with some increase in fibrous tissue and adjacent airway distension." Dr. Graham also noted the presence of acute congestion and edema. He found no asbestos bodies in the lung tissue. Dr. Graham concluded that the miner did not have "any asbestos-related lung disease and that asbestos did not contribute to his lung disease or to his death." Although Dr. Graham stated that "presence of a few small plaques in the visceral pleura is not necessarily indicative of pneumoconiosis," his opinion does not contain any specific statement regarding the presence of coal workers' pneumoconiosis; rather, he appears to focus on the presence or absence of asbestosis.

DR. JEROME KLEINERMAN

Dr. Jerome Kleinerman, who is a board-certified pathologist, reviewed the wet lung tissues, histological slides, and certain medical records and issued a report on May 29, 1999. *Cx.* 3A. He reported 28 and one-half years of coal mine employment, where the miner last worked as a dragline operator. Dr. Kleinerman further noted that the miner was exposed to asbestos for a period of time form operating and maintaining friction brake blocks on the dragline. He reported a 22 to 88 pack year smoking history. Dr. Kleinerman noted that autopsy of the miner was limited to his lungs. Gross examination of the lungs revealed no evidence of pleural plaques or interstitial fibrosis or asbestosis, but there was evidence of chronic bronchitis. Upon review of the slides, Dr. Kleinerman found "nonspecific interstitial fibrosis" and a "rare lesion of simple coal workers' pneumoconiosis." He found no lesions of simple silicosis or complicated pneumoconiosis. There was no significant emphysema present and only minimal black pigment deposition in the lungs. Several sections showed a mild degree of nonspecific interstitial fibrosis, which was present in the subpleural and extra-parenchymal areas. There was no evidence of asbestosis. The right lung revealed pleural thickening and fibrosis over the lateral anterior aspect of the lung surface. Dr. Kleinerman concluded that, based on his review of the medical records, occupational exposure history, chest x-rays, three sets of pulmonary function studies, four sets of arterial blood gas studies, and three carboxyhemoglobin analyses, the miner suffered from simple coal workers' pneumoconiosis. Dr. Kleinerman further opined that the disease was "much too limited and small in extent" to have contributed to the miner's total disability or hastened his death.

With regard to the miner's other testing, Dr. Kleinerman noted that the miner exhibited "chest discomfort" and "shortness of breath" during his 1989 pulmonary function test. He found that all of the values were slightly below normal even though no tracing accompanied the tests.

Dr. Kleinerman found that the test revealed a "mild degree of restrictive lung dysfunction" with only a slight improvement after use of bronchodilators. He further found that the miner's diffusing capacity was "moderately low" and blood gas testing yielded evidence of an oxygen level moderately below normal. Further, he stated that the miner's carboxyhemoglobin level was normal. Subsequent pulmonary function testing in 1991, 1992, 1993, 1995, and 1996 revealed varying obstructive and restrictive impairments. Dr. Kleinerman reported stent placement surgeries in 1995 and 1997. Based on the varying pulmonary function study results, he concluded that the miner was not totally disabled as the result of a respiratory dysfunction. He was, on the other hand, totally disabled due to coronary artery atherosclerosis and narrowing from January 1996 until his death.

DRS. ANUPAMA SHARMA AND VICTOR L. ROGGLI

Dr. Sharma, and Dr. Roggli, who is a Professor of Pathology, reviewed 47 glass slides and issued a report on November 15, 1999. *Dx.* 71. They opined that the slides revealed marked pulmonary edema. There was a "single silicotic nodule" observed in the pulmonary parenchyma and the "hilar lymph nodes show(ed) early fibrotic lesions." Drs. Sharma and Roggli noted that "[n]o typical coal dust macules (were) observed" and there was "minimal dust deposit." They concluded that "[h]istologic evaluation of Mr. Schutt's lung tissue show(ed) minimal changes related to inhalation of silica-containing dust" and stated the following:

> In our opinion, the extensive pulmonary edema and organized thromboemboli are most likely related to Mr. Schutt's cardiac disease, and this is the likely cause of death. Aspiration was most probably an agonal event.

DR. GROVER M. HUTCHINS

Dr. Hutchins is a Professor of Pathology at the Johns Hopkins University and he reviewed the miner's autopsy slides and issued a report on August 20, 1999. *Dx.* 71. Dr. Hutchins noted 29 years of coal mine employment at a strip mine where the miner stopped working in 1989 due to shortness of breath and congestive heart failure. Dr. Hutchins further reported a 21 year history of smoking up to four packs of cigarettes per day, quitting in 1963.

He noted that the miner was on continuous home oxygen therapy since 1996 and that he had undergone multiple angioplasties for obstructive coronary artery disease. Dr. Hutchins also stated that the miner suffered from a myocardial infarction in January 1997 and, on June 2, 1997, he suffered from cardiorespiratory arrest and could not be resuscitated.

Dr. Hutchins found that pulmonary function testing revealed obstructive and restrictive disease and chest x-rays were negative for the presence of pneumoconiosis. He reviewed 52 autopsy slides and made the following findings: (1) "slight amount of subpleural perivascular, and peribronchial coal dust pigment"; (2) no macules, micronodules, macronodules, or lesions of progressive fibrosis; (3) coal workers' pneumoconiosis was not present; (4) there were no asbestos bodies; (5) there were no lesions of silicosis; (6) severe pulmonary edema was present; (7) a mild to moderate degree of centrilobular emphysema was present; (8) chronic bronchitis

was present; and (9) the pleural surface of several slides from the lower lobes showed healed fibrotic reaction and, in one area, it had "produced changes consistent with rounded atelectasis."

Dr. Hutchins concluded that the miner did not suffer from coal workers' pneumoconiosis, but that the miner's "pulmonary or respiratory impairment (was) accounted for by his severe congestive heart failure which arose from his ischemic heart disease." Dr. Hutchins further determined that coal workers' pneumoconiosis did not contribute to the miner's disability or death.

DR. RAPHAEL CAFFREY

Dr. Raphael Caffrey conducted a review of certain medical records and autopsy slides and issued a report on May 3, 1999. *Dx.* 68. He reported 28 years of coal mine employment, where the miner quit on May 19, 1989. Dr. Caffrey further noted a 21 year history of smoking one to four packs of cigarettes per day, quitting in 1963. He found that most of the chest x-ray interpretations did not contain a diagnosis of coal workers' pneumoconiosis. Dr. Caffrey stated that it was his "opinion that (he) definitely (could not) make a diagnosis of coal workers' pneumoconiosis" as the "lung sections actually show only a very minimal amount of anthracotic pigment and/or coal dust."

Dr. Caffrey opined that the miner did suffer from some respiratory impairment at the time of his death, but "that was some eight years after he retired from the mines." As a result, the miner suffered a "cardiac death" given his history of arterosclerotic heart disease and previous myocardial infarction. Dr. Caffrey concluded that years of smoking cigarettes caused the miner's pulmonary impairment, particularly his chronic bronchitis and centrilobular emphysema. Finally, Dr. Caffrey saw no evidence of asbestosis in the lung tissue samples.

DR. STEVEN H. DIKMAN

Dr. Dwight J. Hertz noted, in a letter dated February 13, 2001, that he procured the miner's lung tissue and sent it to Claimant's counsel for referral for examination. *Dx.* 79. Dr. Hertz works in the Department of Pathology at Medcenter One/Q & R Clinic.

Dr. Steven H. Dikman, who is board-certified in anatomical and clinical pathology, performed an autopsy on the miner's lungs and issued a report on November 23, 1998. *Cx.* 2A. Dr. Dikman has been the Associate Attending Physician at the Mount Sinai Hospital since 1980.

He reported 30 years of coal mine employment, where the miner last worked as a dragline operator. Dr. Dikman further noted a 22 year history of smoking cigarettes. He stated that he received a plastic container with two plastic bags labeled left and right lungs. Upon examination, he found "multiple fibrous pleural adhesions" and the "lung markings were coarse with mild environmental pigment deposition."

Dr. Dikman found multiple fibrous pleural adhesions and a small calcified plaque in the medial aspect and linear hyalinized plaque on the lateral aspect. Lung parenchyma revealed emphymatous changes mainly in the upper lobe. Peribronchial lymph nodes contained a

moderate amount of carbon pigment. Sections of the lungs prepared for paraffin blocks showed hyalinized pleural plaquing. Dr. Dikman also found variable interstitial fibrosis and mild to moderate areas of pulmonary emphysema. No asbestos bodies were found in iron stained sections. He found a significant amount of amorphous silica and fibrous and amorphous talc based on electron microscopy, energy dispersive spectroscopy, electron diffraction analysis, and cytocentrifuge light microscopy.

In the right lung, Dr. Dikman observed "multiple areas of pleural fibrosis" and the peribronchial lymph nodes were "prominent and contained moderate amounts of carbon pigment deposits." Overall, Dr. Dikman reported variable interstitial fibrosis and mild to moderate areas of pulmonary emphysema. He stated that "[f]ocal areas of the lung contained small areas of fibrosis with numerous polarizable silicates and mild carbon pigment deposition." He found no asbestos fibers. He noted that "the clinical finding of pleural plaquing was confirmed at autopsy." Dr. Dikman concluded the following:

> Although asbestos was not detected in the tissues examined, the pleural plaquing represents a pneumoconiosis involving the pleura secondary to prior asbestos exposure. The finding of silicate deposition and focal scarring of the lung tissue by microscopic examination was supported by the digestion study showing significant silica deposition in Mr. Schutt's lung and peribronchiolar lymph nodes. This represents a pneumoconiosis attributed to inhaled silicates consistent with coal dust exposure. These changes were superimposed on pulmonary emphysema. It is my opinion, with a reasonable degree of medical certainty, that Mr. Schutt had chronic lung disease with pulmonary emphysema and findings of dust-related pneumoconiosis.

Dr. Dikman further noted the following:

> The finding of silicate deposition and focal scarring of the lung tissue by microscopic examination was supported by the digestion study showing significant silica deposition in Mr. Schutt's lung and peribronchiolar lymph nodes. This represents a pneumoconiosis attributed to inhaled silicates consistent with coal dust exposure. These changes were superimposed on pulmonary emphysema. It is my opinion, with a reasonable degree of medical certainty, that Mr. Schutt had chronic lung disease with pulmonary emphysema and findings of dust-related pneumoconiosis.

DR. SOMSAK KRIENGKAIRUT

Dr. Kriengkairut, one of the miner's treating physicians,[5] conducted an examination of the miner on behalf of the Department of Labor and issued a report dated February 7, 1995. *Dx.* 79. He noted that the miner did not suffer from chest pain, but that he had increasing dyspnea on exertion. Dr. Kriengkrairut reported a 14 year history of smoking two or more packs of cigarettes per day, where the miner quit in 1962, as well as 28 years of above-ground coal mine employment "with close exposure in the pit for about six years, (and) significant exposure to

[5] *See* discussion of treatment records, *infra*

asbestos for seven years." Examination of the lungs revealed decreased air entry, fine rales at both lower lung fields, and occasional rhonchi but no wheezing. Pulmonary function testing did not show significant improvement with the use of a bronchodilator. Indeed, it revealed a slight decrease in the FVC values. Tracings were included. There was a mixed restrictive and obstructive defect due to smoking, coal workers' pneumoconiosis, and asbestosis. Blood gas testing after exercise had to be halted due to increased dyspnea, but not chest pain. Dr. Kriengkairut stated the following:

> It is true that the patient has been working above ground and the majority of the abnormality on x-ray is more on the lower part of the lung, but again, this does not mean that this patient cannot have coal workers' pneumoconiosis because he has been significantly exposed for 28 years above the ground in close space and there is at least 4% of those working above ground who develop coal workers' pneumoconiosis. It is not necessary that coal workers' pneumoconiosis has to be seen only in the upper part of the lung. I believe that this patient's pulmonary pathology is mostly due to underlying coal workers' pneumoconiosis and asbestosis. Smoking might have some impact on this; however, smoking should not be the major part of this patient's pulmonary problem at this time.

Dr. Kriengkairut stated that "[d]yspnea on exertion predominantly (due) to underlying pneumoconiosis/pulmonary asbestosis and less predominantly from chronic obstructive pulmonary disease and smoking." He also stated that the miner suffered from diabetes, exogenous obesity, and atherosclerotic cardiovascular disease. Pulmonary function testing revealed a slight decrease in FVC values from a study conducted three months ago with an FEV1 of 1.86, FVC of 2.69, and MVV of 74 and no significant bronchodilator response. The miner's maximum oxygen consumption was 17.5.

During the exercise test, the miner had a maximum oxygen intake of 17.5, which is low. This limitation, according to Dr. Kriengkrairut was due to a the mixed obstructive and restriction defect." Although the miner experienced shortness of breath and had an abnormal pulmonary response during exercise, he had a normal cardiovascular response to exercise. As a result, Dr. Kriengkrairut pinpointed the miner's disability as one of a pulmonary or respiratory origin, and not of a cardiac origin.

DR. PATRICK STOY

Dr. Patrick Stoy examined the miner, reviewed certain medical records, and issued a report on December 23, 1993. *Dx.* 30. Dr. Stoy is board-certified in internal medicine and allergy and immunology with a sub-specialty in pulmonary disease and critical care. He noted that the miner described experiencing shortness of breath walking 40 feet and that he would awaken at night because of shortness of breath. Dr. Stoy noted that the miner's pulmonary history was negative for asthma, tuberculosis, or other infections besides two bouts of pneumonia. Dr. Stoy reported that the miner smoked one to one and one-half a pack of cigarettes per day for 21 years, quitting in 1963. He also reported the following employment history: (1) started working for Knife River Coal Company in 1959 cleaning coal cars and the tipple; (2) two years were spent as a delivery man driving a car; (3) one year was spent "working

on a 6-inch loader (where) coal would come down a chute and be loaded in the coal cars"; (4) two years of hauling coal; (5) one year as an oiler on the dragline working a coal stripping machine; (6) five to six years on the friction machine where the miner would "take out blocks of asbestos and (change) these blocks when they would wear out"; and (7) for several years the miner drove trucks occasionally and mostly worked on the Prairie Queen, which was a "large electric coal stripping machine which included a 75-yard bucket on it" and the machine was "very clean" such that the miner "did not think he had much dust exposure with this" as the machine "was primarily involved with stripping the dirt and sand off of the earth above the coal and not actually involved with digging the coal us itself." The miner retired in 1987 at the age of 67 years.

Dr. Stoy opined that most of the miner's coal dust exposure occurred in the early years of his employment when he worked at the tipple and drove coal trucks. Dr. Stoy also believed that the miner had some exposure while working the draglines and strippers. The miner wore a mask while working at the tipple, but it fit poorly and when he would cough, the phlegm would be dark or black in color. Dr. Stoy stated the following about the miner's last job:

> At the time of his retirement, his last job involved switching cables and having to walk relatively fast in doing some of this work, and he does describe that he developed some dyspnea with this. Several months earlier, he was involved with doing some shoveling and some work in boxcars and notes that the shoveling activities caused significant dyspnea. They were about to return him to work on the line, as he called it, but he decided to retire instead.

On examination, Dr. Stoy found that the "cardiovascular review of systems (was) completely negative." The heart was normal; there were no gallops and the rhythm was regular. However, the miner did have pulmonary disease. In particular, Dr. Stoy reported the following:

> The chest was barrel shaped, but there was normal chest wall excursion. The breath sounds were distant, particularly in the upper one-half of each lung field on the posterior thorax. There were a few bilateral rales in the lower posterior lung bases, and these did not clear with deep inspiration or coughing.

A chest x-ray revealed emphysema and interstitial fibrosis that was unchanged from November 30, 1993. Pulmonary function testing demonstrated a moderate obstructive airway and mild restrictive lung disease. The diffusion study was "moderately, severely reduced and likely secondary, in part, to the obstructive disease, but interstitial lung disease may also contribute. There was no significant bronchodilator effect."

Dr. Stoy provided the following diagnosis regarding the miner's pulmonary and respiratory condition:

> This individual has moderate obstructive airway disease with a mild to moderate restrictive lung process of undetermined etiology. I do not find evidence to support coal workers' pneumoconiosis by x-ray criteria. He does have findings to

> suggest pleural changes attributable to either asbestos exposure, old infections, or trauma.

Dr. Stoy further opined that the miner's dyspnea was related to "a deconditioned body habitus." In sum, Dr. Stoy found no evidence of coal workers' pneumoconiosis based on his examination, testing, and review of certain medical records. He stated that the miner's "historical exposures to coal mines also do not support coal workers' pneumoconiosis, lacking underground mine exposure and lacking prolonged extensive coal dust exposure as reported in his work history." Dr. Stoy found that the "pulmonary function studies do reveal evidence of reduced diffusion capacity, which would be consistent with some interstitial lung disease." Dr. Stoy concluded:

> Overall, his picture is one of COPD and a restrictive lung disease that is likely due to a combination of pleural thickening, some basilar pulmonary fibrotic changes, obesity, and contributing to his symptoms also is likely a deconditioned body habitus. I do not believe his impairment is due to coal workers' pneumoconiosis.

Dr. Stoy found that the miner's respiratory impairment was attributable to simple obstructive airway disease, which was likely due to his cigarette smoking habits. He concluded that the miner would "likely" be able to perform "some" duties of his last coal mining job, but not strenuous labor. Finally, Dr. Stoy stated that the miner also had "some atherosclerotic cardiovascular disease" that did "not seem to be impairing him.

In a supplemental report dated June 23, 1995, Dr. Stoy stated the following with regard to the miner's condition:

> I would comment that I am in agreement with the findings of these three reviewers, Doctors Castle, Fino, and W.K.C. Morgan. I believe my opinion is similar to their's and that they do bring to further light, some of the discrepancies and possible inadequacies of some of the pulmonary functions studies. It would appear that all of us feel that there is no evidence of significant coal workers' pneumoconiosis.

Dx. 57.

DR. D.M. PFEIFLE

By letter dated December 26, 1991, Dr. D.M. Pfeifle stated the following regarding the miner's condition:

> In answer to your specific questions regarding Benedict Schutt, I see no indication in the record that I ever told the patient his respiratory problems were secondary to work environment nor did I advise him to retire.
>
> I have not seen the patient since December, 1988 and the visits prior to that time were not pulmonary related, primarily his diabetes mellitus and various other minor diagnoses.

Dx. 79. Dr. Pfeifle offered no opinion as to the extent of the miner's disability, if any. He also offered no explanation for his conclusions.

HOSPITALIZATION AND TREATMENT RECORDS

The earliest treatment notes are from March 1976, where the miner complained of chest tightness and wheezing. *Dx.* 4. Examination of the lungs revealed rhonchi bilaterally. The miner was diagnosed with pneumoconiosis based on his symptoms and work history.

Records dated April 4, 1981 indicate that the miner was diagnosed with probable sinusitis and bronchitis as well as a recurring cough. On April 30, 1981, the miner reported dyspnea on exertion, coughing, and some wheezing. Examination revealed an increased AP diameter, increased expiratory phase, and some scattered rhonchi. The miner was diagnosed with sinusitis, bronchitis, and emphysema based on changes noted in his chest x-ray.

On May 27, 1983, examination of the miner's lungs revealed "hyper-resonance in certain parts of the posterior lung" and a chest x-ray revealed advanced chronic obstructive pulmonary disease. The miner was diagnosed with chronic obstructive pulmonary disease, diabetes, obesity, and a history of gout.

On April 25, 1984, the miner complained of a productive cough and occasional wheezing. He had an increased AP diameter and "prolonged expiratory phase and decreased breath sounds" in his lungs. He was diagnosed with chronic obstructive pulmonary disease, hypertension, diabetes, sinusitis, and bronchitis.

On May 23, 1984, the miner's EKG yielded normal results. A chest x-ray was unchanged from one year ago and revealed chronic obstructive pulmonary disease with chronic infiltrates in the left lung.

On April 1, 1986, examination of the miner's lungs revealed "harsh breath sounds." A chest x-ray did not reveal any changes since a May 23, 1986 study. The study showed pleural thickening and fibrosis in the lower left lung. An EKG revealed that an "early transition occurred at the precordium, otherwise (there was) no abnormality." The Cartoid Doppler indicated mild atheromatous disease at the origin of both internal carotoid arteries.

On October 20, 1986, the miner had a cough. Examination of the lungs revealed "decreased breath sounds over the left base with some rales at the left auxiliary area." A chest x-ray showed that the "active process was gone," but that "active infiltrates persist." The miner was diagnosed with remaining bronchitis and resolving pneumonitis.

On November 3, 1986, there was "a lot of rhonchi at the right lung base" and the miner was diagnosed with chronic obstructive pulmonary disease, obesity, and chronic bronchitis.

On July 16, 1987, a chest x-ray revealed chronic obstructive pulmonary disease with interstitial changes. The miner was diagnosed with diabetes, diverticuli of the colon, chronic

obstructive pulmonary disease, probable chest wall pain, and eczema of the external ears. The miner's stress EKG was normal.

Dr. S. Kriengkrairut's treatment notes dated March 17, 1992 are also in the record. *Dx.* 9. He noted that Mr. Schutt had worked in the coal mines and had smoked two packs of cigarettes per day for 22 years. Dr. Kriengkrairut also reported that the miner worked around "brake blocks with asbestos." He diagnosed the miner with chronic obstructive pulmonary disease, coal workers' pneumoconiosis, bilateral pulmonary interstitial lung disease, and asbestos exposure resulting in interstitial lung disease and pleural lung disease.

Q & R Clinic records dated December 16, 1994 provide that a catheterization report did not reveal significant coronary artery disease, but that the miner suffered from shortness of breath. Because the shortness of breath appeared to be due to his lung condition, the miner was referred to a pulmonologist. Notes from the Q & R Clinic dated February 10, 1994 reveal that the miner complained of worsening shortness of breath and wheezing. *Cx.* 4A. The following is stated:

> We advised Mr. Schutt that he appeared to have worsening shortness of breath secondary to his lung problem. From a cardiac standpoint, he had very little coronary artery disease in 1989 and he had at that point a fairly well presented left ventricular function. Since most of his problem appeared to be related to the shortness of breath and since there is no evidence of congestive heart failure, I think that this may be secondary to his chronic obstructive pulmonary disease and asthma.

The miner's Department of Labor-sponsored examination was conducted by Dr. Somsak Kriengkrairut on February 5, 1995. *Dx.* 44. On examination, Dr. Kriengkrairut noted fine rales at the bases of the miner's lungs. He diagnosed atherosclerotic cardiovascular disease with compensated congestive heart failure, chronic obstructive pulmonary disease, coal workers' pneumoconiosis, and pulmonary asbestosis. Dr. Kriengkrairut noted that the miner suffered from coronary artery disease, as documented on the coronary angiogram, but he had "normal ejection faction." He concluded that coal workers' pneumoconiosis, pulmonary asbestosis, chronic obstructive pulmonary disease, and atherosclerotic heart disease contributed to the miner's overall impairment.

On February 7, 1995, Dr. Kriengkrairut's treatment notes reflect that he conducted a pulmonary evaluation and noted that his last pulmonary evaluation of the miner occurred in November 1994. *Cx.* 6A. Dr. Kriengkrairut noted that the miner denied chest pain but that he suffered from worsening dyspnea on exertion. He reported that the miner smoked two or more packs of cigarettes per day for 14 years and he had 28 years of coal mine employment with significant asbestos exposure. Examination of the lungs revealed "decreased air entry with fine rales in both lower lung fields, with occasional rhonchi but no wheezing." Pulmonary function testing yielded evidence of a mixed obstructive and restrictive defect with no bronchodilator response. A chest x-ray demonstrated increased lung markings. He noted that the miner "did not reach an aerobic threshold and stopped exercise due to increasing dyspnea, but no chest pain." Dr. Kriengkrairut found that there was no change in the miner's cardiogram and his maximum

O2 pulse was 16 ml, which was normal and "reflect[ed] normal stroke volume." He diagnosed dyspnea on exertion with deterioration in pulmonary function as seen on blood gas testing at rest and during activity, which was "suggestive of deterioration in restrictive lung disease." Dr. Kriengkrairut concluded that a major component of the miner's pulmonary deterioration was his underlying coal workers' pneumoconiosis and pulmonary asbestosis. Cigarette smoking also contributed to the miner's condition.

Treatment records from the Heart and Lung Clinic dated July 13, 1995 contain diagnoses of pneumoconiosis, asbestosis, chronic obstructive pulmonary disease, and increasing dyspnea on exertion "which has been deteriorating." *Dx.* 78. Fine rales in the lower lung field on the right side with a prolonged expiratory phase were noted.

Dr. Mendoza, a pulmonologist with MedCenter One and Q & R Clinic, issued treatment notes dated August 6, 1996. *Cx.* 8A. He reported 29 years of coal mine employment and asbestos exposure from working with brake linings of heavy equipment. Moreover, he noted a 40 to 50 pack year cigarette smoking history. Dr. Mendoza diagnosed the miner with sleep apnea syndrome "in conjunction with right-sided heart failure and pulmonary hypertension due to his underlying interstitial lung disease and chronic obstructive pulmonary disease. He noted that the chronic obstructive pulmonary disease was "as is seen in coal workers' pneumoconiosis is asthmatic bronchitis, which is demonstrated on his spirometry." Dr. Mendoza further reported a markedly decreased diffusing capacity. Pulmonary function testing revealed a moderate obstruction and mild restriction with no bronchodilator response. Examination of the lungs demonstrated inspiratory rales without wheezing. He diagnosed the miner with coal workers' pneumoconiosis and asbestosis.

MedCenter One records for December 15 and 16, 1996 from Dr. Lunardi are also in the record. *Cx.* 11A. Dr. Lunardi noted that the miner complained of shortness of breath and chest pain. He reported a long history of arteriosclerotic heart disease and angina pectoris as well as coronary artery disease, obesity, diabetes, and hypertension. Dr. Lunardi's discharge diagnoses were pulmonary edema and congestive heart failure, severe thrombocytopenia secondary to Bactrim, history of chronic obstructive pulmonary disease with pulmonary fibrosis, and history of arteriosclerotic heart disease. He noted that the miner was on home oxygen supplementation and had been prescribed Azmacort and Proventil.

Treatment notes dated December 17, 1996 from Dr. F. Addo are in the record. *Cx.* 9A. He noted that the miner's lungs had "harsh vesicular breath sounds" and he diagnosed Bactrim-induced thrombocytopenia and Iron deficiency anemia.

A chest x-ray dated December 17, 1996 revealed "moderate changes of congestive heart failure again." On January 17, 1997, it was noted that both lung fields were resonant to percussion and "breath sounds vesicular with moist basil rales," crackles at the lung bases, and decreased air entry to both lung bases. On January 20, 1997, a few crackles were heard in the lung bases. On January 23, 1997, the miner was diagnosed with severe coronary artery disease.

Dr. Lunardi's February 20, 1997 cardiology notes indicate that the miner had shortness of breath but no chest pain. On March 10, 1997, Dr. Mendoza, a pulmonologist, noted "inspiratory

crackles at both bases" and pulmonary function testing showed some improvement, but that the miner suffered from a mild to moderate obstructive defect. On April 4, 1997, Dr. Lunardi conducted a cardiology examination and noted that there was a probable recurrence of coronary artery disease.

On April 14, 1997, it was noted that the miner had crackles at the lung bases on examination. A portable study revealed "accentuated interstitial densities in the lower lung fields" that had not changed significantly since March 10, 1997.

Notes from Dr. Jan Flattum-Riemers at the Sakakawea Medical Center, dating April 13 and 14, 1997, reveal a few basilar rales in the miner's lungs on examination. *Dx.* 66. A chest x-ray demonstrated cardiomegaly and congestion. The miner's discharge diagnosis was unstable angina, chronic obstructive pulmonary disease, and congestive heart failure.

On April 14, 1997, treating physician Dr. Lunardi states that the miner had a long history of arteriosclerotic heart disease and angina pectoris. *Cx.* 14A. He also reported a history of diabetes mellitus, chornic obstructive pulmonary disease with pulmonary fibrosis, and coronary angioplasty. Dr. Lunardi stated that the miner was a retired dragline operator and that he quit smoking a number of years ago. On examination, he heard a "few crackles in the bases" of the lungs, but found no clubbing, cyanosis, or edema. Moreover, he noted that the peripheral pulses were intact. Dr. Lunardi diagnosed the miner with increasing angina pectoris and stated that impending myocardial infarction would have to be ruled out at some point. He further stated that the miner suffered from anemia.

Medical records from Dr. Lunardi dated April 23, 1997 and May 8, 1997 indicate that the miner underwent an angioplasty. *Dx.* 79. There is a history of arteriosclerotic cardiovascular disease and angina pectoris. Dr. Lunardi noted that the miner underwent "revascularization of the right renal artery" and that he had recurrent congestive heart failure, pulmonary edema, and "critical stenosis of the right renal artery."

From May 6 through May 9, 1997, the miner experienced increasing shortness of breath. *Dx.* 61. Medcenter One records indicate that, in 1989, the miner suffered from a lesion on the right coronary artery. He was placed on medical management and did well until 1995, when he was found to have a critical stenosis in the mid-right coronary artery. Examination of the lungs revealed crackles. The miner complained of increasing shortness of breath. He had very extensive coronary artery disease, significant myocardial dysfunction, and a marked impaired ejection fraction.

On May 9, 1997, the miner was admitted for congestive heart failure. A chest x-ray dated May 6, 1997 revealed pulmonary edema that had developed since a previous study conducted on April 14, 1997. He successfully underwent angioplasty and did well for a year or so, but presented to the clinic again with increasing angina. The miner underwent another angioplasty.

On June 1, 1997, the miner was admitted to Sakakawea Medical Center for right low back pain. *Dx.* 66. His wife arrived at the hospital the next morning and was upset and wanted

her husband transferred to Medcenter One. The attending physician noted that, against her wishes, the miner was not taken by ambulance to Medcenter One; rather, he was taken by car and had stopped at the chiropractor first. Upon leaving the chiropractor, he had to go to the emergency room at Medcenter One.

MedCenter One notes dated June 2, 1997 by Dr. Lange were also admitted into the record. *Cx.* 13A; *Dx.* 67. Dr. Lange noted that the miner was brought to the emergency room by his family and had stopped breathing en route to the hospital. CPR was initiated in the emergency room parking lot, but the miner subsequently died that day. Dr. Lange noted that the miner had "ongoing health problems related to black lung disease and had been on continuous home oxygen since August of 1996." Dr. Lange reported that the family requested a lung autopsy "for legal reasons due to (the miner's) black lung disease." In his notes, Dr. Lange stated that the "[e]xact cause of death is undetermined" and that they were "awaiting the autopsy report, but (he) suspect(ed) that the most likely cause was the cardiac pathology." The emergency room record from Dr. Lunardi on June 2, 1997 reflected that the family consented to an autopsy of the lungs because the "patient has black lung disease." However, Dr. Lunardi opined that the miner passed away as the result of cardiac arrest. *Cx.* 12A.

Discussion of the autopsy evidence

Of the physicians who reviewed the autopsy report and/or slides, the prosector, Dr. Dikman, as well as Drs. Kleinerman and Dolan concluded that there was evidence of simple coal workers' pneumoconiosis in the miner's lungs. Dr. Castle stated that the miner "possibly did have pathologic evidence of coal workers' pneumoconiosis." Dr. Fino stated that he would "assume" that the miner suffered from the disease based on pathologic evidence. On the other hand, Drs. Spagnolo, Sharma, Roggli, Hutchins, and Caffrey concluded that the miner did not suffer from the disease. In addition, Dr. Naeye found only minimal black pigment with associated fibrosis. Indeed, he determined that it was so minimal that he did not diagnose presence of coal workers' pneumoconiosis. However, he did find mild to moderately severe centrilobular emphysema and opined that "smoking has about three times the role of coal mine dust exposure in terms of causing centrilobular emphysema." As will be discussed, this is sufficient to qualify as pneumoconiosis under the regulatory definition at 20 C.F.R. § 718.201 (2001).

It is initially noted that Drs. Spagnolo, Morgan, and Dolan reviewed only the autopsy reports of record. Their opinions are entitled to less weight in determining the nature and extent of disease processes present the miner's lungs because they are not board-certified pathologists. *See, e.g. Livermore v. Amax Coal Co.*, 297 F.3d 668 (7th Cir. 2002) (qualifications of physicians reviewing autopsy evidence should be taken into consideration). Moreover, the lack of such board certification rendered Drs. Spagnolo, Morgan, and Dolan unqualified to review the autopsy slides and, therefore, they merely reviewed the reports of record. Consequently, their reviews of the autopsy evidence are entitled to less weight. *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985) (it is reasonable to assign greater weight to the opinion of a physician who conducts the autopsy over the opinion of a physician who merely reviews the autopsy report); *Fetterman v. Director, OWCP*, 7 B.L.R. 1-688 (1985).

Drs. Sharma, Roggli, Hutchins, and Caffrey are board-certified in pathology and reviewed the autopsy report and slides. They concluded that there were minimal amounts of black pigment with fibrosis in the miner's lungs, but it was too insignificant to qualify as a diagnosis of coal workers' pneumoconiosis under the regulations.

Dr. Dikman, the prosector, found moderate amounts of carbon pigment and multiple areas of pleural fibrosis. He noted that "[t]he finding of silicate deposition and focal scarring of the lung tissue by microscopic examination was supported by the digestion study showing significant silica deposition in Mr. Schutt's lung and peribronchiolar lymph nodes." No other pathologist of record, including Drs. Sharma, Roggli, Caffrey, Hutchins, Naeye and Kleinerman, performed a digestion study. Moreover, these pathologists do not indicate that they performed electron microscopy, energy dispersive spectroscopy, electron diffraction analysis, or cytocentrifuge light microscopy as Dr. Dikman did.[6] While it is not proper to accord greater weight to the prosector's opinion merely because he conducted the autopsy, in this case Dr. Dikman is highly-qualified and he conducted the most thorough examination and testing of the miner's lungs. *Bill Branch Coal Corp. v. Sparks*, 213 F.3d 186 (4th Cir. 2000) (it is improper to accord greater weight to the prosector's opinion solely because he or she reviewed the entire body); *Sabett v. Director, OWCP*, 7 B.L.R. 1-299 (1984) (greater weight may be accorded the opinion that is supported by more extensive documentation). For these reasons, greatest weight is accorded Dr. Dikman's finding of coal workers' pneumoconiosis. Dr. Dikman's opinion is also supported by the report of Dr. Kleinerman. It is noted that, although Dr. Dolan's review of the autopsy evidence is less probative due to his lack of pathological qualifications, his opinion lends further support to the findings of Dr. Dikman.

Dr. Naeye's opinion contains findings that deserve particular consideration.[7] Dr. Naeye observed silica and associated fibrosis in the miner's lungs, but concluded that it was too insignificant to constitute a finding of coal workers' pneumoconiosis under the regulations. On the other hand, he also observed the presence of emphysema. Indeed, Drs. Graham, Hutchins, Caffrey, and Dikman also reported the presence of emphysema. Drs. Dikman and Naeye specifically concluded that the miner had mild to moderate levels of emphysema.

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[6] Dr. Repsher states that the lung digestion study is not used to diagnose coal workers' pneumoconiosis to his knowledge. *Tr.* at 90. Dr. Dolan explained that the study allows the pathologist to "dissolve away the soft tissue and leave only the mineral content of the lung and lymph node." No pathologist of record discredits the study as improper and whether the study is routinely used to diagnose pneumoconiosis is irrelevant. The fact that Dr. Dikman, a pathologist, used this medically acceptable technique along with other techniques to analyze the miner's lungs is appropriate and lends further support to the probative value of his opinion.

[7]Dr. Naeye also mentions a 1.0 centimeter mass in the miner's lung, which contained an insignificant amount of black pigment such that it could not be described as anthracotic. There is no physician of record who diagnoses the presence of complicated pneumoconiosis, including Dr. Dikman, and Claimant has presented no evidence to establish the minimum diameter lesion required on autopsy to qualify as a Category A or greater opacity on chest x-ray. *Smith v. Island Creek Coal Co.*, 7 B.L.R. 1-734 (1985); *Lohr v. Rochester & Pittsburgh Coal Co.*, 6 B.L.R. 1-1264 (1984). Therefore, the record does not support a finding of complicated pneumoconiosis.

Drs. Naeye, Caffrey, and Hutchins posit that the miner suffered from centrilobular emphysema and Dr. Naeye states that smoking played three times the role of coal dust exposure in the development of this condition.

Importantly, however, none of the pathologists, including Dr. Naeye, found evidence of bronchiolitis in the miner's lungs. Dr. Spagnolo explained that it is widely known that a finding of bronchiolitis is required before a diagnosis of centrilobular emphysema is made. In the absence of bronchiolitis, the miner would have suffered from focal emphysema, which is most likely caused by coal dust exposure.

In sum, given the absence of bronchiolitis, it is more likely that the miner suffered from coal-dust-induced focal emphysema. However, even it if was determined that he suffered from centrilobular emphysema, Dr. Naeye's opinion supports a finding that coal dust exposure, along with smoking, would have contributed to this condition.

Thus, the miner suffered from mild simple coal workers' pneumoconiosis as well as a mild to moderate level of emphysema caused, at least in part, by his coal dust exposure. The autopsy evidence supports a finding of pneumoconiosis under 20 C.F.R. § 718.201(a)(2) (2001).

Discussion of the medical opinion evidence

Drs. Dolan, Spagnolo, Morgan, Fino, Castle, Repsher, Kriengkairut, and Stoy are board-certified in internal medicine.[8] They examined the miner or reviewed certain medical records and issued opinions. All of these physicians agree that the miner suffered from a respiratory impairment. Drs. Stoy, Repsher[9], Morgan, and Spagnolo conclude that the miner does not suffer from coal workers' pneumoconiosis. Their reports are entitled to less weight for three reasons.

First, a preponderance of the recent autopsy evidence revealed the presence of mild to moderate levels of coal dust induced respiratory diseases in the miner's lungs. As noted by Dr. Wiot in his deposition, autopsy evidence is the "gold standard" for determining the presence of coal workers' pneumoconiosis. *See also Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). Dr. Stoy did not have the benefit of reviewing and considering the autopsy findings such that his report is less probative. *Church v. Eastern Assoc. Coal Corp.*, 20 B.L.R. 1-8 (1996), *aff'd. in relevant part on recon.*, 21 B.L.R. 1-51 (1997) (it is proper to accord less weight to an opinion that is based on less extensive medical documentation). Drs. Repsher and Morgan relied primarily on the reports of Drs. Caffrey and Hutchins to conclude that there was an insignificant presence of coal dust related macules in the miner's lungs. However, for previously stated

[8] Dr. Pfeifle concluded, in his December 1991 report, that the miner's respiratory "problems" were not caused by his "work environment." However, his opinion is conclusory and is not well-reasoned or well-documented such that it is entitled to no probative value and will not be discussed further. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989) (en banc).

[9] At the hearing, Dr. Repsher stated that there was no definitive evidence of coal workers' pneumoconiosis during the miner's lifetime and that "he may have had very, very mild, simple – simple histologic coal workers' pneumoconiosis." *Tr.* at 92.

reasons, the preponderance of the autopsy evidence revealed mild levels of simple coal workers' pneumoconiosis.

Second, Drs. Spagnolo, Morgan, and Repsher concluded that the miner suffered from smoking-induced emphysema, otherwise known as centrilobular emphysema. For reasons previously set forth in this opinion, it is determined that the miner suffered from focal emphysema caused by coal dust exposure given the failure to find evidence of bronchiolitis in his lungs. However, even if he did suffer from centrilobular emphysema, Dr. Naeye notes that this condition is caused by smoking and, to a lesser extent, by coal dust exposure. Therefore, regardless of the type of emphysema suffered by the miner, coal dust exposure would have caused, or at least contributed to, its development. Given the more accurate findings on autopsy, the opinions of Drs. Spagnolo, Morgan, and Repsher are less probative.

Third, Drs. Spagnolo, Morgan, and Repsher attributed the miner's respiratory impairment to his smoking history on grounds that pulmonary function testing produced a purely obstructive defect. In *Cornett v. Benham Coal Co.*, 227 F.3d 569 (6th Cir. 2000), the Sixth Circuit held that such opinions were not well-reasoned. In particular, the court stated the following under the facts before it:

> Each of the three doctors unfavorable to Cornett reported that his respiratory problems were caused by his smoking habit *only*. If this is so, Cornett's ailments do not qualify as statutory pneumoconiosis. *See* 20 C.F.R. § 718.201. But, of the three, only Dr. Fino attempted to explain his rationale for completely excluding Cornett's exposure to coal dust as an aggravating factor. Dr. Fino attributed Cornett's obstructive lung disease to cigarette smoking because, in his opinion, the pulmonary function tests (revealing pure obstruction) were not consistent with 'fibrosis as would be expected in simple coal workers' pneumoconiosis.' What the ALJ did not consider in his opinion is that, although 'fibrosis' is generally associated with 'medical' pneumoconiosis, it is not a required element of the broader concept of 'legal' pneumoconiosis. (citation omitted). The legal definition does not require 'fibrosis' but instead requires evidence that coal dust exposure aggravated the pulmonary condition.

Similarly, in this case, the physicians who cite to the miner's pure obstruction on pulmonary function testing to conclude that his respiratory impairment does not arise from coal dust exposure are only addressing the concept of "medical", and not "legal", pneumoconiosis.

On the other hand, Drs. Dolan and Kriengkairut reasonably conclude that the miner suffered from a coal dust related respiratory impairment.[10] Their reports are supported by a preponderance of the autopsy evidence of record. In addition, the miner consistently exhibited symptoms of crackles, rales, rhonchi, and/or wheezing during his treatment or examinations by

[10] Dr. Castle opined that the miner "probably" suffered from coal workers' pneumoconiosis based on the autopsy evidence. Similarly, Dr. Fino stated that, based on the autopsy evidence, he would "assume" that the miner had coal workers' pneumoconiosis.

pulmonologists. Also, the obstructive impairment on pulmonary function testing was irreversible. This is consistent with the presence of coal workers' pneumoconiosis, which may be a purely obstructive impairment, and which is progressive and irreversible. 20 C.F.R. § 718.201(a)(2) and (c) (2001); *Lane Hollow Coal Co. v. Lockhart*, 137 F.3d 799, 803 (4th Cir. 1992) (pneumoconiosis is irreversible). Finally, the most recent blood gas study of record, which post-dates other studies by at least one and one-half years, yielded qualifying values. *Morgan v. Bethlehem Steel Corp.*, 7 B.L.R. 1-226 (1984) (such evidence may "bear upon the existence of pneumoconiosis insofar as test results indicate" the presence of any disease process and, by implication, the presence of disease arising out of coal mine employment).

Drs. Fino and Castle opined that clinically significant coal workers' pneumoconiosis would cause reduced diffusing capacity. In 1993, Dr. Fino stated that the miner's diffusing capacity values were normal, which ruled out the presence of the disease. However, in his 1995 report, Dr. Fino noted that the miner suffered from an oxygen transfer abnormality. He posited that, based on the autopsy reports of record, there was not enough intrinsic lung disease to account for the abnormality. In his December 1996 report, Dr. Repsher noted that the miner's low diffusing capacity "suggest(ed) significant underlying emphysema." Dr. Morgan attributed the miner's reduction in diffusion capacity "to his former habit of cigarette smoking which caused his emphysema along with the fact that his cardiac output was reduced."

Dr. Castle also found that the miner suffered from reduced diffusing capacity, but stated that "when it was corrected for volume, it was essentially normal, or at the lower limit of normal." However, upon review of Dr. Mendoza's 1996 testing, he specifically corrected diffusing capacity for alveolar volume and still found a "significant loss of gas exchanging surface." This is consistent with the most recent blood gas testing of record conducted during the same time period, which yielded qualifying values. Dr. Castle noted that either a fibrotic process, such as coal workers' pneumoconiosis, or a "significantly destructive process," such as emphysema, will cause a reduction in diffusion capacity. Thus, the miner's reduced diffusing capacity lends further support to a finding that he suffered from coal dust-induced respiratory impairments.

In this case, the autopsy evidence establishes that the miner suffered from mild simple coal workers' pneumoconiosis as well as mild to moderate emphysema arising, at least in part, from his coal dust exposure. The presence of both mild coal workers' pneumoconiosis and mild to moderate emphysema arising, at least in part, from coal dust exposure in the miner's lungs would reasonably contribute to the reduction in his diffusing capacity.

Consideration of whole record–pneumoconiosis established

Upon review of the evidence as a whole, it is determined that the miner suffered from coal workers' pneumoconiosis under 20 C.F.R. §§ 718.201 and 718.203(b) (2001); *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000). Although the chest x-ray evidence was preponderantly negative for presence of the disease, the most probative autopsy evidence establishes that the miner suffered from coal workers' pneumoconiosis as well as mild to moderate levels of emphysema arising, at least in part, form coal dust exposure. This is supported by the treatment records and probative examining physicians' opinions of record,

which reveal progressively worsening shortness of breath, a reduced diffusing capacity, recent qualifying blood gas testing, and routine symptoms of rales, rhonchi, crackles, and/or wheezing on examination of the lungs. Inconsistencies in pulmonary function testing are reasonably attributed to variation in effort, as testing notes indicate that the miner was often hindered by dyspnea and coughing during these examinations. It is noteworthy, however, that he exhibited no significant response to bronchodilators on pulmonary function testing, which is consistent with an irreversible condition such as coal workers' pneumoconiosis. In sum, the probative evidence of record establishes that the miner suffered from coal workers' pneumoconiosis under 20 C.F.R. §§ 718.201 and 718.203(b) (2001).

VI
Total Disability Due To Pneumoconiosis

Benefits are provided under the Act for or on behalf of miners who are totally disabled due to pneumoconiosis. 20 C.F.R. § 718.204(a) (2000).[11] The regulations at § 718.204(c) provide the following five methods to establish total disability: (1) qualifying pulmonary function studies; (2) qualifying blood gas studies; (3) evidence of cor pulmonale with right-sided congestive heart failure;[12] (4) reasoned medical opinions; and (5) lay testimony.[13] 20 C.F.R. § 718.204(c) (2000).

Consideration of pulmonary function studies

Total disability may be established through a preponderance of qualifying pulmonary function studies. The quality standards for pulmonary function studies are located at 20 C.F.R. § 718.103 (2001) and require, in relevant part, that each study be accompanied by three tracings, *Estes v. Director, OWCP*, 7 B.L.R. 1-414 (1984), that the reported FEV1 and FVC or MVV values constitute the best efforts of three trials, and, for claims filed after January 19, 2001, a flow-volume loop must be provided. The administrative law judge may accord lesser weight to those studies where the miner exhibited "poor" cooperation or comprehension. *Houchin v. Old Ben Coal Co.*, 6 B.L.R. 1-1141 (1984); *Runco v. Director, OWCP*, 6 B.L.R. 1-945 (1984). To be qualifying, the regulations provide that the FEV1 and either the MVV or FVC values must be equal to or fall below those values listed at Appendix B for a miner of similar gender, age, and height.[14] The following pulmonary function studies are in the record:

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[11] The pre-amendment definition of "total disability" will be utilized as the miner's claim was filed prior to January 19, 2001. 65 Fed. Reg. 69,930, 69,935 (Dec. 15, 2003); *National Mining Ass'n. v. Dep't. of Labor*, 292 F.3d 849 (D.C. Cir. 2002).

[12] There is no evidence of cor pulmonale with right-sided congestive heart failure such that this method of establishing total disability will not be discussed further.

[13] The Board holds that a judge cannot rely solely upon lay evidence to find total disability in a living miner's claim. *Tedesco v. Director, OWCP*, 18 B.L.R. 1-103 (1994).

[14] The Board holds that, if the record contains substantial differences in the heights recorded on the studies of record, a factual finding must be made regarding the claimant's actual height. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221 (1983). Based upon the record in this case, it is determined that Claimant is 66.5 inches in height.

Exhibit No./ Date of Study	Coop/Comp./ age/height	FEV1/ FVC/ MVV	Br?	Tracings?	Qualify?	Comments
Dx. 36 06-08-89	?/? 66 years/ 66.5"	2.17/ 3.15/ 89 2.25/ 3.28/ --	No Yes	yes yes	no no	mild obstructive defect acutely responsive to bronchodilator. Suggest underlying emphysema. "The nature of the abnormality in the diffusing capacity and diffusing coefficient suggests a reduction in lung volume that can be seen in interstitial lung disease"
Dx. 28 03-17-92	good/good 69 years/ 68"	2.11/ 3.00/ --	No	yes	no	Mixed obstructive, restrictive defect. Restrictive defect seems to be the major component of dyspnea. "Increasing dyspnea on minimal exertion seems to be much worse clinically and objectively on the pulmonary function tests and exercise test on today's visit."
Dx. 29 11-30-93	good/good 71 years/ 67"	1.95/ 2.73/ --	No	yes	no	"Unable to get 3 consistent efforts due to patient's shortness of breath; "patient very short of breath"
Dx. 29 12-16-93	good/good 71 years/ 67"	1.37/ 1.83/ – 1.48/ 1.91/ --	no yes	yes yes	yes yes	"Good effort with patient having excessive coughing"; "mild restrictive disease combined with moderate obstructive disease. The diffusion study is moderately severely reduced, likely secondary in part to the obstructive disease but interstitial lung disease may contribute. Bronchodilator effect is not significant."

Dx. 79 02-07-95	good/good 72 years/ 67"	1.86/ 2.69/ 74 1.85/ 2.63/ 74	no yes	yes yes	no no	
Dx. 61 08-06-96	?/? 73 years/ 67"	1.83/ 2.83/ --	No	yes	no	No significant improvement with bronchodilator. Mild restrictive ventilatory defect, moderate obstructive ventilatory defect. Diffusing capacity decreased to 42% consistent with alveolar capillary block and interstitial lung disease. Reduced DLCO/VA (correcting diffusing capacity for alveolar volume) suggests significant loss of the gas exchanging surface.
Dx. 61 08-22-96	?/? 73 years/ 67"	2.18/ 3.25/ --	No	yes	no	Mild obstructive ventilatory defect. Improvement from 8-6-96.
Dx. 61 11-18-96	?/? 74 years/ 66"	1.58/ 2.26/ --	No	yes	no	Moderate restrictive defect.
Dx. 61 03-10-97	?/? 74 years/ 66"	2.04/ 3.44/ --	No	yes	no	Mild-moderate obstructive ventilatory defect.

Based upon the foregoing, the miner has not established total disability pursuant to § 718.204(c)(1) of the regulations. The preponderance of the pulmonary function testing reveals low, but not totally disabling, values. In particular, the most recent testing of record, which was indicative of the miner's capabilities, produced non-qualifying results.

However, non-qualifying pulmonary function testing does not preclude a finding of total disability based on qualifying blood gas testing. Pulmonary function and blood gas testing measure different types of impairment. In *Tussey v. Island Creek Coal Co.*, 982 F.2d 1036, 1040-41 (6th Cir. 1993), the court held that pulmonary function and blood gas testing "may consistently have no correlation since coal workers' pneumoconiosis may manifest itself in different types of pulmonary impairment." *See also Sheranko v. Jones and Laughlin Steel Corp.*, 6 B.L.R. 1-797, 1-798 (1984).

Consideration of blood gas studies

Total disability may be established by qualifying blood gas studies under § 718.204(c)(2) (2001). In order to be qualifying, the PO2 values corresponding to the PCO2 values must be equal to or less than those found at the table at Appendix C. The following blood gas studies are in the record:

Exhibit No.	Date	Resting (R)/ Exercise (E)	Altitude (ft)	PCO2	PO2	Qualify?
Dx. 57	06-09-89	R	0-2,999	36.2	60.8	Yes
Dx. 57	03-17-92	R E	0-2,999	42.1 40.1	76.2 74	No No
Dx. 61	12-16-93	R E	0-2,999	37.5 38	66.2 63	No No
Dx. 45	02-07-95	R	0-2,999	44.7	64.2	No
Dx. 46[15]	02-10-95	R	0-2,999	40.8	53.6	Yes
Dx. 37	07-31-96	R	0-2,999	38.6	59.2	Yes

The most recent blood gas test, dated July 31, 1996, yielded qualifying values and supports a finding that the miner was totally disabled. Given the fact that pneumoconiosis is progressive, it is proper to accord greatest weight to the July 1996 test, which post-dates all other blood gas testing by one and one-half to seven years. *Thorn v. Itmann Coal Co.*, 3 F.3d 713 (4th Cir. 1993); *Schretroma v. Director, OWCP,* 18 B.L.R. 1-17 (1993). As a result, the most recent blood gas study testing establishes that the miner was totally disabled.

Consideration of medical opinion evidence

The final method by which Claimant can establish total disability due to pneumoconiosis is through medical opinion evidence wherein a physician has exercised reasoned medical judgment based on medically acceptable clinical and laboratory diagnostic techniques to conclude that the miner's respiratory or pulmonary condition prevents him from engaging in his usual coal mine employment or comparable employment. 20 C.F.R. § 718.204(c)(4) (2000).

Initially, Claimant has the burden of establishing the exertional requirements of his usual coal mine employment. *Onderko v. Director, OWCP*, 14 B.L.R. 1-2 (1989). Once a claimant establishes that he is unable to perform his usual coal mine employment, a *prima facie* case for total disability exists and the burden shifts to the party opposing entitlement to prove that the

[15] By report dated February 10, 1995, Dr. Timothy Kennedy concluded that the February 10, 1995 blood gas study at exercise was "valid and abnormal." *Dx.* 43.

claimant is able to perform comparable and gainful work. *Taylor v. Evans and Grambrel Co.*, 12 B.L.R. 1-83, 1-87 (1988).

Prior to a hearing in this matter, Claimant passed away and was, therefore, unable to testify as to the job duties of his last coal mine employment as a dragline operator. As a result, the undersigned will rely in part on the history provided by the miner to Dr. Stoy, the limited testimony of his son, Robert Schutt, at the hearing as well as on the definition of "dragline operator" set forth in the *Dictionary of Occupational Titles*, 4th ed. (DOT). The DOT is compiled and published by the U.S. Department of Labor's Employment and Training Administration and contains the official job descriptions and exertional requirements for numerous jobs. The job of "dragline operator," listed as 850.683-018, is defined as follows:

> Operates power-driven crane equipped with dragline bucket, suspended from boom by cable to excavate or move . . . coal: Drives machine to work site. Moves hand levers and depresses pedals to rotate crane on chassis and position boom above excavation point, to raise and lower boom, to lower bucket to material, to drag bucket toward crane to excavate or move material. May direct workers engaged in placing blocks and outriggers to prevent capsizing of machine when lifting heavy loads. May be designated according to type of chassis or power unit as Crawler-Dragline Operator (any industry); Gasoline-Dragline Operator (any industry); or material excavated as Clay Hoister (cement); Walking-Dragline Operator (any industry).

Id. at 889. The definition trailer designates the occupation's strength as "M." According to Appendix C of the DOT, this means that the position requires "medium work," which is defined as follows:

> Exerting 20 to 50 pounds of force occasionally, and/or 10 to 25 pounds of force frequently, and/or greater than negligible up to 10 pounds of force constantly to move objects. Physical Demand requirements are in excess of those for Light Work.

Id. at 1013. In his report, Dr. Stoy noted that the miner stated that, "[a]t the time of his retirement, his last job involved switching cables and having to walk relatively fast in doing some of this work, and he does describe that he developed some dyspnea with this."

The miner's son, Robert Schutt, works for Dakota Westmoreland, formerly known as Knife River Coal Company. *Tr.* at 21. Mr. Schutt testified that he knew the job duties required of his father as a dragline operator for the company and he worked for the company during essentially the same period of time. *Tr.* at 39. As a dragline operator, the miner worked in an enclosed cab. *Tr.* at 48. The cab had heating in the winter, but no air conditioning until years after the miner worked as an operator. *Tr.* at 49. Therefore, a miner would open up all the windows on the cab in the summer to let the air flow. *Tr.* at 49.

Comparing the exertional requirements of his last coal mining job with the physical limitations demonstrated on this record, it is determined that Claimant has established that the

miner was totally disabled under § 718.204(c)(4) through a preponderance of the medical opinion evidence of record.

Except for Dr. Stoy, all of the physicians of record conclude that the miner was totally disabled.[16] Dr. Spagnolo stated that the miner's disability arose from coronary artery disease and smoking-induced lung disease. He did not find that the miner suffered from clinical coal workers' pneumoconiosis but stated that, even if it was present, "it had to be in such an extremely limited or minor condition as to not have any impact on (Mr. Schutt's) pulmonary function or lung function."

Dr. Morgan opined that the miner suffered from a non-disabling respiratory impairment arising from his smoking history, but that the miner's obesity, age, and cardiac problems left him totally disabled. Dr. Morgan also found that the miner did not suffer from clinical coal workers' pneumoconiosis and noted that there was an "insignificant" presence of coal macules on autopsy and the macules were "perfectly harmless and (did) not produce any impairment."

Dr. Fino concluded that the miner had "oxygen transfer abnormalities" due to his tobacco abuse and the miner was totally disabled from his last job. Dr. Castle stated that the miner did not retain the respiratory capacity to return to his previous coal mining job, but the impairment was related only to his smoking history.

Dr. Repsher concluded that the miner suffered from totally disabling coronary artery disease and smoking-induced chronic obstructive pulmonary disease. Dr. Repsher opined that the miner did not suffer from clinical pneumoconiosis such that he was not impaired by the disease during his lifetime.

Dr. Kleinerman did find the presence of clinical coal workers' pneumoconiosis, but stated that pulmonary function testing did not indicate a respiratory impairment; rather, the miner was totally disabled due to coronary artery artherosclerosis and narrowing from January 1996 until his death.

Dr. Naeye opined that the miner did not suffer from clinical or legal coal workers' pneumoconiosis as his mild to moderately severe emphysema would not be sufficient to impair the miner from a respiratory standpoint. Dr. Hutchins stated that the miner did not suffer from clinical or legal coal workers' pneumoconiosis and his "respiratory impairment (was) accounted for by his severe congestive heart failure which arose from his ischemic heart disease." Dr. Caffrey concluded that the miner did not suffer from clinical or legal coal workers'

[16] At the time of his December 1993 report, Dr. Stoy concluded that the miner suffered from a respiratory impairment "likely due to his smoking habits" and that he would be "likely" able to perform "some" duties of his last coal mining job as a dragline operator. First, it is noted that Dr. Stoy's equivocal assessment constitutes an insufficient basis upon which to assess whether the miner was totally disabled. *Parsons v. Black Diamond Coal Co.*, 7 B.L.R. 1-236 (1984) (an equivocal opinion regarding disability may be accorded less weight). Second, Dr. Stoy's opinion is based on his 1993 examination and, therefore, does not constitute an assessment of the miner's deteriorating physical condition in later years. *Gillespie v. Badger Coal Co.*, 7 B.L.R. 1-839 (1985). It is further noted that Dr. Pfeifle's report is not probative regarding the issue of total disability as he specifically decline to render an opinion on the issue.

pneumoconiosis, but his respiratory impairment arose from smoking-induced chronic bronchitis and centrilobular emphysema.

Drs. Dolan and Kriengkairut opined that the miner suffered from totally disabling respiratory impairment arising, in part, from his coal dust exposure.

The undersigned has found the presence of *clinical* coal workers' pneumoconiosis as well as *legal* pneumoconiosis, *i.e.* existence of mild to moderate levels of emphysema arising, at least in part, from coal dust exposure. Drs. Naeye, Caffrey, Hutchins, Spagnolo, Repsher, and Morgan concluded that the miner's respiratory impairment, to the extent that he suffered from such an impairment, was due to his tobacco abuse, and not coal dust exposure. Consequently, these physicians' opinions are based on a premise in disagreement with the undersigned's findings on this record.

Because Drs. Naeye, Caffrey, Hutchins, Spagnolo, Morgan, and Repsher did not find the presence of clinical or legal pneumoconiosis contrary to the undersigned's findings, their opinions are entitled to little weight with regard to the etiology of the miner's disability. *Scott v. Mason Coal Co.*, 289 F.3d 263 (4th Cir. 2002); *Toler v. Eastern Assoc. Coal Co.*, 43 F.2d 109 (4th Cir. 1995).

Drs. Kleinerman, Fino, Repsher, and Castle did state that the miner suffered from coal workers' pneumoconiosis based on the autopsy data of record. Drs. Kleinerman, Repsher, and Fino opined that there was an insufficient level of coal workers' pneumoconiosis on pathology to account for the oxygen transfer abnormalities revealed by the miner's testing. Similarly, Dr. Castle opined that "based upon the sparsity of findings in the pathologic specimens and the lack of physiologic impairment associated with coal workers' pneumoconiosis," the "process was so minimal as to have not caused him any impairment during life . . .." However, the preponderance of evidence of record supports a finding that the miner did not suffer from only "minimal" or "insignificant" coal workers' pneumoconiosis. It is apparent that Drs. Kleinerman, Fino, Repsher, and Castle restricted their causation opinions to addressing the impact of *clinical* pneumoconiosis. Indeed, Dr. Castle testified that the miner did not suffer from *legal* pneumoconiosis during his deposition. Their opinions regarding causation are accorded little probative value because Drs. Kleinerman, Fino, Repsher, and Castle failed to consider the combined effects of the miner's mild simple coal workers' pneumoconiosis and mild to moderately severe coal dust induced emphysema. It is reasonable to conclude that the combined effects of these conditions on the miner's respiratory and pulmonary systems was not "minimal" or "insignificant." In fact, Drs. Fino, Castle, and Repsher concluded that the miner's respiratory impairment contributed to his disability, although they opined that this impairment arose from tobacco abuse contrary to a preponderance of the evidence in this record.

On the other hand, Drs. Dolan and Kriengkairut persuasively opine that coal workers' pneumoconiosis contributed to the miner's total disability. Dr. Kriengkairut conducted a comprehensive examination of the miner in 1995. Two important observations were made at this point in time. First, Dr. Kriengkairut observed that the miner experienced dsypnea on exertion and that, during an exercise test, the miner's maximum oxygen consumption was 17.5. Dr. Dolan testified at his deposition that "this exercise level shows that (the miner) wasn't even able

to reach his maximum predicted heart rate because of shortness of breath." As a result, Dr. Kriengkairut found that the miner exhibited an abnormal pulmonary response to exercise. Second, Dr. Kriengkairut conducted testing of the miner's cardiovascular system and found that it responded normally to exercise. From this, Dr. Kriengkairut reasonably concluded that the miner's disability was of a pulmonary or respiratory origin, not of a cardiac origin.

Dr. Dolan agreed that the miner's coal workers' pneumoconiosis contributed to his total disability. He closely tracked the miner's treatment and hospitalization records and noted that the miner developed a respiratory ailment, which progressively worsened over the years. Specifically, Dr. Dolan stated the following in his January 1997 report:

> (The miner's) treating doctors have recorded severe respiratory symptoms, such as inability to mow even with a power mower (1991), having to stop to breathe three times walking from the parking lot to the clinic (1992), dyspnea on minimal exertion (1993), gradually worsening dyspnea on exertion (1995). He currently uses oxygen by nasal cannula. The latest pulmonary exercise test I have reviewed, dated 2/10/95, showed severe exercise limitation. He could only exercise at low exertion level for 6 minutes, dropping his oxygen saturation from 92 to 85. At an oxygen saturation of 90% (equaling a PO2 of approximately 60), most people will have the sensation of being short of breath. After his very modest exertion, with an oxygen saturation of 85% (PO2 of 53.6), he probably felt very uncomfortably short of breath. According to the physician's comments, his condition was worsening with time. On the basis of the symptoms noted by his physician and corroborated by the exercise testing, Mr. Schutt cannot perform any reasonable work and presently seems to require supplemental O2 just for activities of daily living.

Dr. Lunardi, in treatment notes dated December 1994, noted that a heart catheterization revealed no significant coronary artery disease such that the miner's shortness of breath was more due to his lung condition and he should be analyzed by a pulmonologist. The undersigned finds that Dr. Dolan's opinion is well-reasoned and well-documented.[17]

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[17] At one point, Dr. Dolan also cited to the American Medical Association (AMA) guidelines to support his opinion that the miner suffered from a totally disabling respiratory impairment. In its brief, Employer maintains the following:

> Dr. Brian Dolan, the claimant's expert, not a pulmonary specialist, concluded that there was a Class III impairment of the whole person using the AMA guidelines. (citation omitted). Dr. Repsher, who edited the second through fifth editions of the AMA's *Guidelines for the Evaluation of Permanent Impairment* chapters on pulmonary diseases explained that the exercise studies failed to reveal a pulmonary impairment but showed a class III cardiac impairment. Dr. Repsher explained the pulmonary criteria could not be used to evaluate impairment for individuals with severe heart disease or individuals in heart failure. Instead, the cardiac section is utilized. (citation omitted).

Id. at 23-24. The record does not, however, establish that the miner suffered from severe heart disease or heart failure in February 1995, when the miner exhibited "severe exercise limitation." To the contrary, Dr. Dolan specifically noted that there was no evidence that the miner suffered from any congestive heart failure at that point in time. Indeed, Dr. Kriengkairut stated, during his February 1995 testing, that the miner exhibited a "normal cardiovascular response to exercise," but his pulmonary response was "abnormal."

Upon review of the record as a whole, it is apparent that the miner developed progressively worsening and well-documented respiratory symptoms, starting around 1975. These symptoms worsened, but there was no concomitant evidence of impairing cardiac problems at the time. Dr. Stoy noted, during his 1993 examination, that "cardiovascular review of (the miner's) systems (was) completely negative." In 1995, Dr. Kriengkairut found that the miner exhibited a normal cardiovascular response to exercise, but his pulmonary response was abnormal. It is reasonable that, by this time, the miner was no longer able to perform the duties of dragline operator. Dr. Kriengkairut noted a severely reduced diffusing capacity and that the miner could not complete the exercise portion of the examination due to shortness of breath. It is evident that the miner would not be able to switch cables, walk quickly, exert force with his hands or feet, or perform the other moderately strenuous duties of his last job.

By 1996, Dr. Dolan noted that the medical records reveal continued worsening of the miner's respiratory condition (to the point that the miner used supplemental oxygen) and the development of cardiac problems. In 1996, the miner was hospitalized for coronary artery disease, which, as noted by Dr. Dolan, was successfully treated with angioplasty. Moreover, during a 1996 hospitalization, Dr. Dolan found that a thallium test revealed right ventricular decompensation. By May 1997, he was hospitalized for congestive heart failure and, in June 1997, he suffered respiratory arrest and could not be resuscitated.

It is reasonable from the documented deterioration in the miner's health through the treatment and hospitalization records, that he was totally disabled, at least in part, to his respiratory impairment. As previously noted, coal dust exposure contributed to the miner's respiratory impairment. Therefore, Claimant has established by a preponderance of the evidence that the miner was totally disabled due, at least in part, to coal workers' pneumoconiosis and is entitled to benefits arising from the miner's lifetime claim.

VII
Establishing death due to pneumoconiosis

Benefits are provided under the Act for survivors of miners who died due to pneumoconiosis. 20 C.F.R. § 718.205 (2001). The regulations at § 718.205 require competent medical evidence which (1) establishes that the miner died due to pneumoconiosis; or (2) that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or (3) that the presumption of § 718.304 is applicable.[18] Moreover, "[p]neumoconiosis is a 'substantially contributing cause' of a miner's death if it hastens the miner's death." 20 C.F.R. § 718.205(c)(5) (2001).

[18] Lay evidence provisions at § 718.204(c)(5) are inapplicable to this survivor's claim because it was filed after January 1, 1982. *See also Gessner v. Director, OWCP*, 11 B.L.R. 1-1, 1-3 (1987). Moreover, as previously noted, Claimant has not established that the miner suffered from complicated pneumoconiosis, thus that method of finding total disability will not be discussed.

Initially, physicians' opinions regarding the cause of death, which are premised on a finding of no clinical or legal pneumoconiosis, are entitled to little weight.[19] Consequently, the opinions of Drs. Naeye, Caffrey, Hutchins, Sharma, Roggli, Spagnolo, and Morgan will be accorded little weight with regard to the cause of death because they failed to diagnose clinical or legal pneumoconiosis. *See Scott, supra; Toler, supra.*

Drs. Kleinerman, Fino, Repsher, and Castle conclude that the miner died due to his cardiac problems and/or a respiratory impairment related solely to his tobacco abuse. These physicians conclude that the miner's coal workers' pneumoconiosis was so insignificant that it could not have hastened his death. However, as previously noted, the undersigned is not persuaded that the combined effects of mild simple coal workers' pneumoconiosis and mild to moderately severe emphysema, arising in part from coal dust exposure, found on autopsy had a *de minimus* effect on the miner's condition during his life and at the time of his death. Because Drs. Kleinerman, Fino, and Castle did not find that the miner's emphysema was related, at least in part, to his coal dust exposure, their opinions as to the cause of death are less probative.

On the other hand, Dr. Dolan persuasively explains that the miner's coal dust-induced respiratory ailments hastened his death. In particular, he noted that the miner's respiratory symptoms were present from 1975 until 1995, without data to support the presence of any concomitantly impairing cardiac disease. By 1996, the miner used supplemental oxygen. Dr. Dolan emphasized that the miner's respiratory ailments progressively worsened over time based on a review of the miner's treatment and hospitalization records. As a result, he disagreed with the conclusions of Dr. Lange, who is a cardiologist, on the miner's death certificate:

> I was at a loss to explain why (Dr. Lange) wrote on the death certificate that the cause of death was cardiac arrest with arrhythmia due to arteriosclerotic cardiovascular disease when there's no indication that he had any further information which would sway him more toward cardiac than pulmonary.

Given the lengthy and progressively severe respiratory impairment documented in the miner's treatment and hospitalization records, the undersigned finds Dr. Dolan's opinion the most probative as to the cause of death. It is more likely than not that the coal dust-induced respiratory impairments hastened the miner's death, which involved respiratory failure. No other physician of record examined the miner's medical history as closely as Dr. Dolan nor did any other physician explain the progress of the miner's conditions culminating in his total disability and death as persuasively as Dr. Dolan. Dr. Dolan is a highly-qualified physician who is board-certified in internal medicine, preventative medicine, and occupational medicine. Moreover, he has a Master's degree in Public Health.

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[19] The *Certificate of Death* lists the cause of death as cardiac arrest with arrhythmia due to arteriosclerotic cardiovascular disease. Dr. Lange, the cardiologist who completed the *Certificate,* does not explain his conclusions. In fact, in his emergency room report, Dr. Lange indicated that he was uncertain as to whether the miner's death was cardiac or pulmonary in nature. As a result, the death certificate does not constitute probative evidence as to the cause of the miner's death in this case. *Risher v. Office of Workers' Compensation Programs*, 940 F.2d 327, 331 (8th Cir. 1991); *Smith v. Camco Mining, Inc.*, 13 B.L.R. 1-17 (1989); *Addison v. Director, OWCP*, 11 B.L.R. 1-68 (1988).

Based on the foregoing, Claimant has established by a preponderance of the evidence that the miner's death was hastened by his respiratory impairment due, at least in part, to coal dust exposure.

VIII
Onset of Benefits

Entitlement to miner's benefits

Claimant is entitled to benefits commencing on the date the medical evidence first establishes that he became totally disabled due to pneumoconiosis or, if such a date cannot be determined from the record, the month in which the miner filed his claim which, in this case, is February 1992. 20 C.F.R. § 725.503 (2001); *Carney v. Director, OWCP*, 11 B.L.R. 1-32 (1987); *Owens v. Jewell Smokeless Coal Corp.*, 14 B.L.R. 1-47 (1990). It is noteworthy that the date of the first medical evidence of record indicating total disability does not establish the onset date; rather, such evidence only indicates that the miner became totally disabled at some prior point in time. *Tobrey v. Director, OWCP*, 7 B.L.R. 1-407, 1-409 (1984); *Hall v. Consolidation Coal Co.*, 6 B.L.R. 1-1306, 1-1310 (1984).

Upon review of the record in this case, it is determined that the onset date is February 1995, based on Dr. Kriengkairut's observations and testing during his examination of the miner. The only physician to examine the miner prior to Dr. Kriengkairut was Dr. Stoy. In December 1993, Dr. Stoy found that the miner suffered from a respiratory impairment, most likely due to his tobacco abuse, and that he could perform some of his former job duties. Dr. Stoy did not diagnose the presence of legal or clinical coal workers' pneumoconiosis.

On the other hand, Dr. Kriengkairut found that the miner suffered from a disabling respiratory impairment arising, in part, from coal dust exposure, as supported by his exercise testing, and this impairment contributed to the miner's total disability. Dr. Kriengkairut also diagnosed the presence of coal workers' pneumoconiosis. He noted that exercise testing of the miner had to be halted due to increased dyspnea, but not chest pain. By August 1996, the miner needed to use supplemental oxygen for daily activities, and subsequent testing and examinations revealed continued deterioration of the miner's condition. There is insufficient medical data preceding Dr. Kriengkairut's examination upon which to determine the precise date on which the miner became totally disabled. Objective testing prior to February 1995 yielded non-qualifying values. On the other hand, there is medical documentation to support a finding that the miner's condition progressively worsened after Dr. Kriengkairut's examination. Therefore, it is reasonable to conclude that benefits on the living miner's claim should be awarded from February 1995.

Entitlement to survivor's benefits

Where the claimant is an eligible survivor of the miner and entitled to benefits under the Act, as in this case, such benefits must be paid beginning with the month of the miner's death but, in no instance, before January 1, 1974. 20 C.F.R. § 725.503(c). The survivor in this claim is entitled to benefits from June of 1997, the month in which the miner died. Accordingly,

ORDER

IT IS ORDERED that the claims for benefits pursued by Agnes Schutt, as the widow of and on behalf of deceased miner Benedict Schutt, are granted and benefits on the living miner's claim are payable commencing as of February 1995 until May 1997, the month preceding the miner's death.

IT IS FURTHER ORDERED that survivor's benefits are granted and benefits are payable commencing as of June 1997, the month in which the miner died.

IT IS FURTHER ORDERED that, on or before March 31, 2004, Claimant's counsel, David Thompson, shall file, with this Office and with opposing counsel, a petition for a representatives' fees and costs in accordance with the regulatory requirements set forth at 20 C.F.R. § 725.366. Counsel for the Director and Employer shall file any objections with this Office and with Claimant's counsel on or before April 16, 2004. It is requested that the petition for services and costs clearly state (1) counsel's hourly rate and supporting argument or documentation therefor, (2) a clear itemization of the complexity and type of services rendered, and (3) that the petition contains a request for payment for services rendered and costs incurred before this Office **only** as the undersigned does not have authority to adjudicate fee petitions for work performed before the district director or appellate tribunals. *Ilkewicz v. Director, OWCP*, 4 B.L.R. 1-400 (1982).

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Thomas M. Burke
Associate Chief Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this Notice of Appeal must also be served on Donald S. Shire, Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C. 20210.